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The Northeast Regional Environmental Impact Study

Reference Document for the Health Effects of Air Pollution

November 1981

Argonne National Laboratory, Argonne, Illinois

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ACRONYMS AND ABBREVIATIONS

AAQS	Ambient Air Quality Standards
ACGIH	American Converence of Governmental and Industrial Hygienists
AHH	Aryl hydrocarbon hydroxylase
ATPase	Adenosine triphosphatase
CO	Carbon monoxide
CoHb	Carboxyhemoglobin
CHESS	Community Health and Environment Surveillance System
BaP	Benzo(a)pyrene
BS	British Smoke (filter method)
CHO	Chinese Hamster ovary
EIS	Environmental Impact Statement
ESP	Electrostatic precipitator
HC	Hydrocarbons
HGPRT	Hypoxanthine-guanine phosphoribosyl transferase
HV	High-volume
KI	Potassium-iodide
MCDCE	Manhattan College Department of Chemical Engineering
MMAD	Mass median aerodynamic diameter
NAAQS	National Ambient Air Quality Standards
Na-K ATPase	Sodium-potassium-activated adenosine triphosphatase
NASN	National Air Surveillance Network
NEPA	National Environmental Policy Act
NIOSH	National Institute of Occupational Safety and Health
NO	Nitric oxide
NO_2	Nitrogen dioxide
O_3	Ozone
PAHs	Polycyclic aromatic hydrocarbons
PAN	Peroxyacetyl nitrate
PNAs	Polynuclear aromatic hydrocarbons

POM	Polycyclic organic matter
OSHA	Occupational Safety and Health Administration
SO_2	Sulfur dioxide
SS	Suspended sulfates
TLV	Threshold Limit Value
TSP	Total suspended particulates
USDOE	U.S. Department of Energy
USEPA	U.S. Environmental Protection Agency

ABSTRACT

The U.S. Department of Energy (USDOE) is assessing the potential for cumulative and interactive environmental impacts associated with the proposed conversion to coal of up to 42 powerplants in the Northeast Region of the United States under the Powerplant and Industrial Fuel Use Act of 1978 (Pub. L. 95-620). USDOE's Northeast Regional Environmental Impact Study provides analysis in four interrelated areas: (1) air quality, (2) solid waste disposal, (3) fuel supply and the transportation of fuel and solid waste, and (4) health effects. This technical reference document is a summary of literature on health effects related to emissions from coal burning in general; it does not specifically address the health effects associated with the conversion of the 42 powerplants under consideration. Areas of concern and differing views that result in uncertainty are identified. Graphics and tabular data display the health data with environmental data and standards for comparison. Both criteria and noncriteria pollutants are included.

1. INTRODUCTION, OBJECTIVE AND COVERAGE OF THE REPORT

1.1 INTRODUCTION

The proposed action to be assessed in the Northeast Regional analysis is the cessation of the use of oil and natural gas as primary energy sources in up to 42 powerplants in the northeastern United States. The objective of the proposed action is, in consonance with the purposes of the Powerplant and Industrial Fuel Use Act of 1978 (Pub. L. 95-620), to minimize or eliminate oil consumption in as many of these units as possible. Among the functions that the U.S. Department of Energy (USDOE) performs under the Act are negotiating voluntary conversions and working with those utilities subject to the authorities of the Act to encourage them to pursue conversions.

USDOE can encourage fuel switching away from oil by providing technical analyses of the effects of fuels conversion. The Northeast Regional Environmental Impact Study provides this type of analysis in four interrelated areas: (1) air quality; (2) solid waste disposal; (3) fuel supply and the transportation of fuel and solid waste; and (4) health effects. A separate technical task report is being prepared in each of these areas, and will serve both as a general reference document and as a technical reference for the environmental impact statements issued under the Fuel Use Act. This report is the technical task report on health effects.

The primary purpose of the Northeast Regional Environmental Impact Study is to assess and document the potential for cumulative and interactive environmental impacts associated with the conversion of multiple generating stations in the Northeast. The 42 facilities included in the study (see Table 1.1) were selected because they were considered by the President's Coal Commission to be coal-capable. This Commission originally compiled a list of 117 generating stations that were considered capable of using coal. This list was reduced by USDOE using the criteria of eliminating (1) all units over 25 years of age and (2) stations with an aggregate capacity of less than 100 megawatts. The size and age criteria focused attention on powerplants that had the greatest potential for oil displacement and economic benefits, and on units having the longest remaining useful life. The overall area addressed by the Northeast Regional analysis is the macroregion defined by Maryland to the South and Maine to the North. The facilities are distributed over 10 states, with a majority of them clustered in the New York-New Jersey-Connecticut tri-state region (Fig. 1.1). In addition, in the area of air quality, specific attention is focused on four subregions centering around Boston, New York City, Philadelphia, and Baltimore. The state of Vermont generally has been excluded from the study, as it contains none of the subject utility boilers, nor is it considered a candidate area for waste disposal. The depth and breadth of coverage of this regional document will be sufficient to provide a data base and analysis for site-specific environmental analysis as well as a broader perspective of the overall impacts on the Northeast Region, as described in the Northeast Regional Environmental Impact Statement (NEREIS) (USDOE 1981). Detailed treatment is not included in the study, nor are aspects more relevant to site-specific environmental impact statements. Instead, generic issues that are cumulative or interactive on a regional basis are emphasized. This approach conforms to the intent of the National Environmental Policy Act (NEPA) in general, and to the Council on Environmental Quality Regulations on implementing NEPA procedures in particular, as the documents provide the middle tier of a three-tiered approach to impact assessment. The first tier is the published Final Programmatic Environmental Impact Statement for the Fuel Use Act (USDOE 1979), and the Revised Programmatic Environmental Impact Statement for the Energy Supply and Environmental Coordination Act (Federal Energy Administration 1977). The NEREIS is the middle tier and the final tier is composed of the site-specific environmental impact statements.

1.2 OBJECTIVE AND COVERAGE

This Task Report on Health Effects summarizes what is known about the health effects of the air pollutants associated with coal combustion, primarily in fossil-fueled powerplants. This document relates to coal burning in general; it is not specific enough to address directly the conversion of the 42 plants under consideration. This health-effects literature review can, however, serve as a basis for addressing the site-specific impacts of powerplant fuel conversions from oil to coal and can play an important role in decisions concerning cost-effective pollution control measures. This report is not intended to provide an independent review of the scientific data behind air pollutant criteria standards but is intended to summarize current knowledge. Where existing reviews define areas of concern or where differing views result in

Table 1.1. Facilities Included in the Northeast Regional Environmental Impact Study

State/Facility	Unit Number
Connecticut	
Bridgeport Harbor	3
Devon	7,8
Middletown	1,2,3
Montville	5
Norwalk Harbor	1,2
Delaware	
Edge Moor	1,2,3,4
Maine	
Mason	1,2,3,4,5
Maryland	
Brandon Shores	1,2
Crane	1,2
Riverside	4,5
Herbert A. Wagner	1,2
Massachusetts	
Canal	1
Mt. Tom	1
Mystic	4,5,6
New Boston	1,2
Salem Harbor	1,2,3
Somerset	6
West Springfield	3
New Hampshire	
Schiller	4,5,6
New Jersey	
Bergen	1,2
Burlington	7
Deepwater	7,8,9
Hudson	1
Kearny	7,8
Sayreville	4,5
Sewaren	1,2,3,4
New York	
Albany	1,2,3,4
Arthur Kill	2,3
Danskammer Point	1,2,3,4
E. F. Barrett	1,2
Far Rockaway	4
Glenwood	4,5
Lovett	3,4,5
Northport	1,2,3,4
Oswego	1,2,3,4
Port Jefferson	1,2,3,4
Ravenswood	3
Pennsylvania	
Cromby	2
Schuylkill	1
Southwark	1,2
Springdale	7,8
Rhode Island	
South Street	12

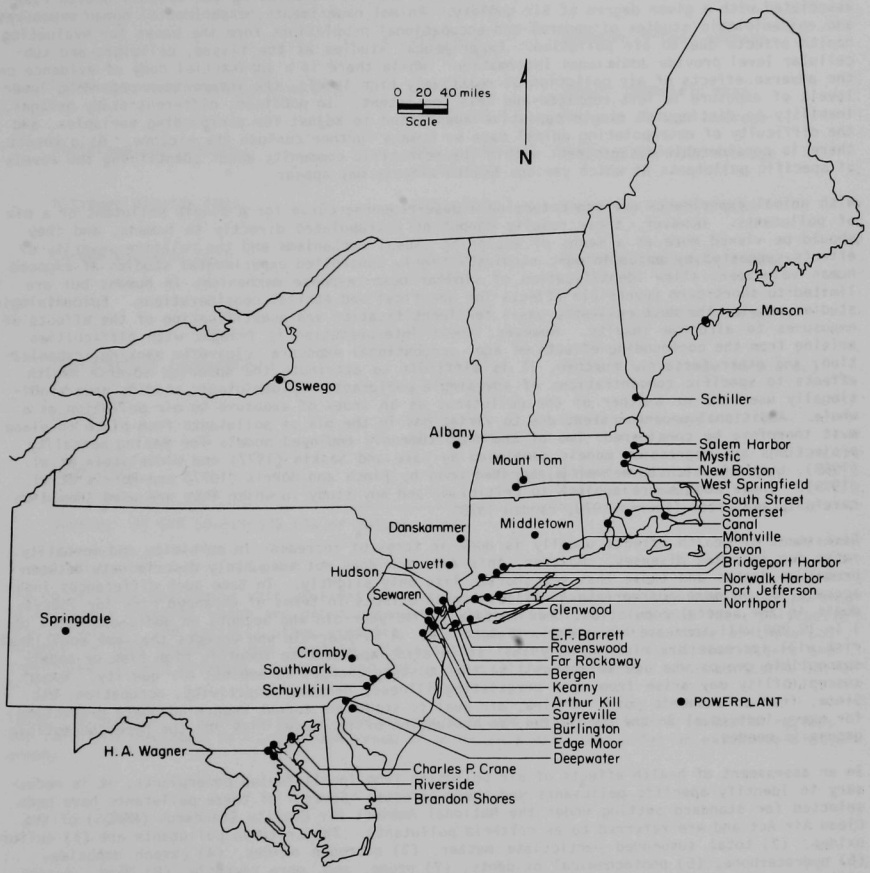

Fig. 1.1. Facilities Inluded in the Northeast Regional Environmental Impact Study

considerable uncertainty, such concerns or uncertainties are, however, clearly identified. Graphics and tabular data display the health data with environmental data and standards for quick comparison.

The evaluation of the health risks from air pollutants involves (1) identifying specific health effects, (2) quantifying these effects at various pollutant concentrations, (3) estimating how many people are exposed at these concentrations, and (4) calculating the overall health risk associated with a given degree of air quality. Animal experiments, experimental human exposures, and epidemiologic studies of general and occupational populations form the bases for evaluating health effects due to air pollution. Experimental studies at the tissue, cellular, and subcellular level provide additional information. While there is a substantial body of evidence on the adverse effects of air pollution at relatively high levels, the information regarding lower levels of exposure is less complete and less consistent. In addition, different study designs, inability to distinguish single causative agents and to adjust for confounding variables, and the difficulty of extrapolating animal data to humans further confuse the picture. As a result, there is considerable disagreement within the scientific community about quantifying the levels of specific pollutants at which various health effects may appear.

With animal experiments one can determine a dose-response curve for a single pollutant or a mix of pollutants. However, these results cannot be extrapolated directly to humans, and they should be viewed more as a means of exploring causal mechanisms and the relative severity of effects suggested by epidemiologic studies. Highly controlled experimental studies of exposed human volunteers allow identification of similar dose-response mechanisms in humans but are limited to short-term reversible effects for practical and ethical considerations. Epidemiologic studies provide the most realistic basis for identification and quantification of the effects of exposures to airborne insults. However, their interpretation is fraught with difficulties arising from the confounding effects of age, occupational exposure, cigarette smoking, urbanization, and other factors. Further, it is difficult to attribute the observed adverse health effects to specific concentrations of any single pollutant. Epidemiologic studies have traditionally used one or another of the pollutants as an index of exposure to air pollution as a whole. Additional uncertainties, due to variations in the mix of pollutants from place to place must therefore be considered. Two of the most commonly employed models for making mortality projections are regression models developed by Lave and Seskin (1977) and Winkelstein et al. (1968), both of which have been elaborated upon by Finch and Morris (1977) and Morris et al. (1979). These models are subject to criticism, and any study in which they are used should be carefully evaluated (Ferris 1978; Landau 1978).

Assessment of health effects usually is done in terms of increases in morbidity and mortality rates for selected diseases. However, this approach does not adequately discriminate between premature deaths and those that may shorten life only slightly. To take such differences into account, it is more appropriate to assess health effects in terms of enhanced risk for individuals in the general population. For instance, a 10-year-old who accepts an additional risk of 1 in 10,000 will increase his overall risk by 38%. A 50-year-old who accepts the same additional risk will increase his risk by only 1%. It is also important to identify high risk or hypersusceptible groups who are more sensitive than most to changes in ambient air quality. Hypersusceptibility may arise from age, preexisting illness, genetic sensitivity, occupation, etc. Since, from an economic point of view, air quality standards cannot ensure adequate protection for every individual in the population, an evaluation of increased risk for the hypersusceptible groups is needed.

In an assessment of health effects of air pollution from fossil-fueled powerplants, it is necessary to identify specific pollutants and their effects. Several of these pollutants have been selected for standard setting under the National Ambient Air Quality Standards (NAAQS) of the Clean Air Act and are referred to as criteria pollutants. The criteria pollutants are (1) sulfur oxides, (2) total suspended particulate matter, (3) nitrogen oxides, (4) carbon monoxide, (5) hydrocarbons, (6) photochemical oxidants, (7) ozone, and, more recently, (8) lead. A significant number of experimental and epidemiological studies have been undertaken to serve as the basis for the standards developed under the NAAQS (Table 1.2). This body of scientific information has received extensive review (Ferris 1978; American Thoracic Society/American Lung Association 1978; Holland et al. 1979). Most published reviews range from those in support of the current criteria pollutant standards as being appropriate to those in which the standards are considered to be too stringent. This report is not intended to provide an independent review of the scientific data behind the criteria pollutant standards.

USEPA has proposed to rescind the unused air quality standard for hydrocarbons as a pollutant class because it has no value under the Clean Air Act. The standard (0.24 ppm, measured from 6 to 9 a.m.) was not intended to be enforceable but was issued as a guide for meeting the national air standard for ozone (Barber 1981).

Table 1.2. U.S. Primary Air Quality Standards

Pollutant	Standard	Averaging Time
Sulfur dioxide (SO_2)	80 $\mu g/m^3$ (0.03 ppm)	Annual arithmetic mean
	365 $\mu g/m^3$ (0.14 ppm)	Maximum 24-hour average
Total suspended particulates (TSP)	75 $\mu g/m^3$	Annual geometric mean
	260 $\mu g/m^3$	Maximum 24-hour average
Photochemical oxidants	235 $\mu g/m^3$ (0.12 ppm)	Maximum one-hour average
Nitrogen dioxide (NO_2)	100 $\mu g/m^3$ (0.05 ppm)	Annual arithmetic mean
Carbon monoxide (CO)	10 $\mu g/m^3$ (9 ppm)	Maximum eight-hour average
	40 $\mu g/m^3$ (35 ppm)	Maximum one-hour average
Hydrocarbons (nonmethane)	160 $\mu g/m^3$ (0.24 ppm)	Three-hour average (6-9 a.m.)
Ozone	235 $\mu g/m^3$ (0.12 ppm)	One-hour average
Lead (Pb)	1.5 $\mu g/m^3$	Calendar quarter maximum Arithmetic mean

Source: 40 CFR 50:523-525 (As of July 1, 1980).

The noncriteria pollutants with greatest potential for health damage include respirable particulates, trace elements which are often associated with particulates, radionuclides, polycyclic organic matter, and possibly other organics. Although the relevant literature on these pollutants is less organized and complete, the data available on each class of pollutants are summarized in this document in graphs and tables in much the same format as the criteria pollutants.

Table 1.3 provides a quick guide to key data collections on air pollution in health reviews. The reference sections following each group of pollutants discussed offer an extensive bibliography.

Table 1.3. Index to Key Air Pollution Health Reviews

Subject of Review	Author (Date)
General	U.S. Office of Technology Assessment (1979) Ferris (1978) American Thoracic Society/American Lung Association (1978) Stern (1977) National Academy of Engineering (1975) World Health Organization (1972) Wilson et al. (1981)
Sulfur oxides	National Academy of Sciences (1979) National Research Council (1975a) Colucci (1976) U.S. Dept. of Health, Education and Welfare (1970a) USEPA (1974a,b) Rall (1974)
Particulates	Holland et al. (1979) National Research Council (1979) Perera and Ahmed (1978) National Academy of Engineering (1975) National Research Council (1972a) U.S. Dept. of Health, Education and Welfare (1969)
Nitrogen oxides, photochemical oxidants, and hydrocarbons	Colucci and Simmons (1978) USEPA (1978a) National Research Council (1977a) Morrow (1975) National Institute for Occupational Safety and Health (1972a) U.S. Dept. of Health, Education and Welfare (1970c, 1971) USEPA (1971) Brezonik (1978)
Ozone	USEPA (1978b) National Research Council (1977b) U.S. Dept. of Health, Education and Welfare (1970b)
Carbon monoxide	National Academy of Sciences (1977) USEPA (1979)
Lead	USEPA (1977) National Research Council (1980)
Radionuclides	McBride et al. (1978) Eisenbud and Petrow (1964) National Research Council (1972b)
Trace elements	Lim (1979) Sunderman (1978) Friberg et al. (1977) Fishbein (1976) National Institute for Occupational Safety and Health (1972b, 1973a,b, 1974, 1975, 1976) National Academy of Sciences (1975, 1976) National Research Council (1974, 1975b)
Polycyclic organic matter	Santodonato et al. (1979) National Research Council (1972a)

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Brezonik, P.O. (Chairman). 1978. Nitrates: An Environmental Assessment. Panel on Nitrates of the Coordinating Committee for Scientific and Technical Assessment of Environmental Pollutants. National Academy of Sciences, Washington, D.C.

Colucci, A.V. 1976. Sulfur Oxides: Current Status of Knowledge. EPRI EA-316. Electric Power Research Institute, Palo Alto, Calif.

Colucci, A.V., and W.S. Simmons. 1978. Nitrogen Oxides: Current Status of Knowledge. EPRI EA-668. Electric Power Research Institute, Palo Alto, Calif.

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2. OVERVIEW OF FINDINGS

2.1 INTRODUCTION

The impacts of coal-combustion-process releases on human health are dependent on many factors (National Academy of Engineering 1975). The body's response to these impacts may differ according to the age and condition of the victim, the nature of the specific pollutant, and the duration of exposure. Health effects associated with air pollution from combustion include acute health effects, chronic respiratory disease, exacerbation of asthma, children's respiratory disease, aggravated heart-lung disease, and air-pollution-associated mortality. Table 2.1 is a summary of some of the gaseous and particulate pollutants from coal combustion, their general toxicity, major sources of emissions, and applicable environmental standards.

The present state of knowledge does not permit a quantification of the absolute health effects associated with various air pollutants. However, their health effects can be assessed on a comparative basis and furthermore, for at least some pollutants, threshold levels below which effects are not discernible (or are negligible compared to other factors) have been demonstrated. Most published reviews of the scientific evidence behind the NAAQ standards range from those in support of the standards as being appropriate to those in which at least some standards are considered to be conservative. On the basis of these standards (which are correlated with data on health effects), other known facts about health effects, and source/exposure-related information, it is fair to state the following conclusions regarding the relative health effects of fossil-fueled powerplants.

The contribution of coal combustion to levels of carbon monoxide (CO), hydrocarbons (HC), and lead is negligible compared to that of transportation and the relative health risks from these pollutants attributable to coal combustion are not significant. It should also be noted that trends for photochemical oxidants (one constituent of which is hydrocarbons) and carbon monoxide, the most troublesome pollutants in U.S. cities, have improved significantly during the period 1973-76 in most cities, due to stringent controls on automobile emissions (Council on Environmental Quality 1978). The HC, CO, and lead emissions are expected to decline even further in coming years (MITRE Corp. 1978). The trend is the opposite for overall emissions of nitrogen oxides. Control of nitrogen oxide emissions from mobile sources is more than offset by increased emissions from coal-fired utilities and boilers which presently account for almost 40% of the nitrogen oxide emissions. Compared with sulfur dioxide (SO_2) and total suspended particulates (TSP), emissions of nitrogen oxides are relatively serious because with increasing use of coal, the upward trend in nitrogen oxide emissions (ambient concentrations are already high) is likely to continue unless appropriate control technologies are developed. Meeting current National Ambient Air Quality Standards (NAAQS) for nitrogen oxides of 0.05 ppm or 100 $\mu g/m^3$ (annual arithmetic mean concentration) will be more difficult in the large population concentrations like the Northeast. Increases in emissions of nitrogen oxides are also likely to affect oxidant levels.

There has been a declining trend in SO_2 emissions (the major source of which is coal combustion), due primarily to fuel switching, the enforcement of relatively strict standards for old facilities, and even more stringent controls for new sources. One study indicates that increasing use of coal will reverse this trend to some extent, and that SO_2 emissions are likely to remain essentially the same as in 1975 (MITRE Corporation 1978). SO_2 concentrations per se do not present a serious problem from the point of view of health effects. The main concern is over the conversion of SO_2 to sulfates (SO_4), which currently are suspected to be causing the health effects associated with SO_2/particulate pollution. Sulfates also contribute to the acid rain problem. On a very localized level, decreasing emissions of sulfur oxides do not always result in decreased sulfate (or even SO_2) concentrations. This apparent anomaly generally is attributed to the fact that sulfates can be transported over much greater distances than SO_2 and remain in the atmosphere much longer. Sulfate concentrations in the northeast quadrant of the U.S. generally are the highest in the country and have not declined in recent years in spite of reductions in corresponding SO_2 levels. The relationship between SO_2 levels and transformation products is not yet well understood.

Overall particulate emissions also exhibit a declining trend and are projected to decrease further (MITRE Corporation 1978). Unlike sulfur oxide emissions, particulate emissions originate

Table 2.1. Some Gaseous and Particulate Substances from Coal Combustion[a]

Substance[b]	Toxicity		Sources of Pollution	Environmental Standards	Comments
	Acute	Chronic			
PARTICULATES					
Total suspended particulates	With SO_2 in episode conditions contributes to mortality and morbidity.	Pulmonary irritation, chronic obstructive and restrictive lung disease.	Soil erosion, natural volcanoes and fires, industrial activity, fossil fuel combustion: coal and oil, secondary atmospheric conversion of gaseous compounds.	260 $\mu g/m^3$ 24-hr max.; 75 $\mu g/m^3$ ann. avg.	A very broad class, undifferentiated by particle size or chemical composition.
Sulfates	Increased respiratory disease; breathing difficulty in asthmatics.	Respiratory disease and increased mortality suspected.	Conversion of SO_2 to sulfates in the atmosphere; therefore primary sources are SO_2 emissions from coal and oil combustion. Smelters, kraft paper mills, sulfuric acid plants also produce sulfates. Natural sources--H_2S emissions, volcanoes, sea salt.		
Nitrates, nitrites	Increases infant susceptibility to lower respiratory infection due to conversion of nitrates to nitrites.	May combine with amines to form carcinogenic nitrosamines; also mutagenic and teratogenic. Nitrites a direct animal carcinogen.	Conversion of NO_2 to nitrates and nitrites in the atmosphere; therefore primary sources are NO emissions from fossil-fuel combustion, fertilizer production, munition production, chemical plants, auto and industrial emissions.		
Organic matter	Unknown for many compounds. Specific toxicity for others.	Long-term is potentially carcinogenic and and mutagenic.	Fossil fuel direct and indirect use--combustion, refining, plastics, tars, coking, chemical production.		Higher concentrations likely to be associated with nondirect combustion of fuels.
Arsenic (oxide forms)	Effects large to small depending on form and route of exposure; rarely seen.	Carcinogen, and teratogenic cumulative poison.	Weathering; mining and smelting; coal combustion; pesticides; detergents.	0.05 mg/L drinking water.	
Beryllium	Short-term poison at high concentrations, especially toxic by inhalation.	Long-term systemic poison at low concentrations; carcinogenic in experimental animals.	Industrial; combustion of coal, rocket fuels.	0.01 $\mu g/m^3$ hazardous air pollutant.	
Cadmium	Very toxic at high concentrations to animals and aquatic life. Toxic by all routes of exposures.	Possible carcinogen, cumulative poison; associated with hypertension, cardiovascular disease, kidney damage.	Weathering; mining and and smelting, especially of zinc; iron and steel industry; coal combustion; urban runoff; phosphate fertilizers.	0.010 mg/L drinking water; 40 $\mu g/L$/day proposed effluent standards (withdrawn).	Chronic cadmium poisoning resulting in illness and death occurred in Japan, where cadmium mobilized by mining contaminated daily diet. Margin of safety--measured levels of cadmium in renal cortex compared to threshold for renal dysfunction--is low: 4 to 12.5.

Table 2.1 (concluded)

Substance	Toxicity: Acute	Toxicity: Chronic	Sources of Pollution	Environmental standards	Comments
Chromium	Hexavalent form most harmful; skin and respiratory tract irritant.	Carcinogenic; workers engaged in manufacture of chromium chemicals have incidence of lung cancer; no evidence of risk in nonoccupational exposure.	No chromium now mined in U.S. Emissions from industrial processes, including electroplating, tanning, dyes; coal combustion.	0.05 mg/L drinking water.	
Mercury	Methyl mercury and mercury fumes very toxic; other forms of variable toxicity.	Methyl mercury very toxic, cumulative poison; affects central nervous system.	Weathering; volcanoes; mining and smelting; industrial; pharmaceuticals; coal combustion; sewage sludge; urban runoff; fungicides.	0.002 mg/L drinking water; maximum of 2300 g mercury in emissions from stationary sources; 20 µg/L/day proposed effluent standard (withdrawn).	Environmental pollution leading to contamination of fish and shellfish caused illness and death in Japan; contamination of fish in U.S. has caused closure of waters to commercial fishing.
Selenium	Soluble compounds highly toxic.	Probable carcinogen; also essential for life.	Natural; mining and smelting; industrial process; coal combustion.	0.01 mg/L drinking water.	Interacts with other metals, increasing or decreasing toxicity.
GASES					
Sulfur dioxide	Increased respiratory impairment--morbidity and mortality--in combination with particulates.	Increased respiratory disease and decreased respiratory function with particulates.	Sulfur contained in fossil fuels, smelters, volcanoes.	365 µg/m^3 24-hr max.; 80 µg/m^3, ann. mean.	Coal cumbustion presently represents between 60 and 70% of U.S. SO_2 emissions.
Nitrogen dioxide	Increased respiratory infections.	Changes suspected in lung function; emphysema.	Nitrogen fixation in high temperature combustion, and from nitrogen contained in fossil fuels; coal, oil, gasoline combustion.	100 µg/m^3 ann. mean.	Organically bound fuel nitrogen is a more important component for coal NO emissions than for the other fossil fuels.
Carbon monoxide	Behavioral changes, nausea, drowsiness, headaches, coma, death.	Increased risk of coronary heart disease--arterial sclerosis suspected.	Incomplete combustion of fossil fuels; coal, oil, gas, gasoline combustion.	40 mg/m^3 1-hr max.; 10 mg/m^3 8-hr max.	
Ozone	Increased respiratory infection, eye irritation, headaches, chest pain, impaired pulmonary function.	Unknown	Photochemical reactions involving hydrocarbons, nitrogen oxides, and other other compounds in lower atmosphere; reaction of atomic oxygen and oxygen in upper atmosphere.	160 µg/m^3 1-hr max.	
Aromatic hydrocarbons	Fatigue, weakness, skin paresthesias (> 100 ppm).	Irritation, leukopenia and anemia. Certain compounds are mutagens and carcinogens.	A broad class of compounds naturally evolved from organic material, and from the evaporation and combustion of fossil fuels and other organic industrial chemicals.	Nonmethane HC, 160 µg/m^3 3-hr.	Higher concentrations likely from less efficient and and smaller boiler operation. Higher concentrations possible proximate to coal conversion facilities. Standard designed for photochemical oxidant control.

From U.S. Office of Technology Assessment (1979).

[a]This table is provided to indicate some of the substances that are potential environmental hazards along with some information on each regarding toxicity, sources, standards, etc. It is not to be interpreted as definitive.

[b]The substance listed is not necessarily the form in which it becomes a potential environmental threat. In some cases the oxide or some metabolite, rather than the substance itself, is the potential threat.

from a variety of sources, including the structural materials industry (crushed stone), the iron and steel industry, and several other nonenergy sources. These sources account for most of the particulate pollution in the U.S. Utilities and boilers, which constituted about 30% of the particulate emissions in 1975, are expected to contribute even less in the future, because of the installation of electrostatic precipitators and baghouses on boilers. This will lead to even fewer violations of the TSP standards (generally considered to be adequate), which already are rare. Large TSP emissions and high ambient particulate concentrations as measured by air quality monitoring stations are highly correlated in the East (MITRE Corporation 1978).

In summary, an integrated assessment of health risks from various criteria pollutants attributable to coal combustion indicates that the primary concerns should be for emissions of nitrogen oxides and transformation products of SO_2 such as sulfates, from both the health effects and exposure points of view. Most concerns arising from noncriteria pollutants have to do with the toxicity and suspected carcinogenicity of many trace elements and organics and their ready availability via the large surface area provided by fine particles upon which they concentrate during coal combustion. This availability allows intimate contact with body fluids as well as the respiratory system (U.S. Office of Technology Assessment 1979).

The relationships between exposures to specific agents and health effects are summarized below.

2.2 THE SULFUR OXIDE/PARTICULATE COMPLEX

The sulfur oxide and particulate complex produced during the combustion of sulfur-containing fossil fuels is the major component of air pollution released from coal-fired powerplants. In 1975, 62% of all sulfur oxide emissions in the U.S. came from coal-fired electric utilities (MITRE Corporation 1978).

Historically, the association between sulfur dioxide (SO_2) pollution and adverse health effects was manifested by acute, high-exposure air pollution episodes that resulted in increased mortality and respiratory illness (Ferris 1978). Subsequent studies (Amdur 1957, 1959; Amdur and Underhill 1968; Alaire et al. 1972) indicated that SO_2 by itself is not a potent pulmonary irritant and could not be the primary causative agent. The apparent reason for the low potency is that inhaled SO_2 is almost completely absorbed in the upper airways and does not reach the lung. Sulfur dioxide penetrates into the lungs only when it is adsorbed onto the surface of respirable particulate matter (approximately 2 μ diameter or less).

More studies of human health effects of sulfur oxides and particulates have been conducted than for the other components of air pollution. Both short-term acute air pollution episodes and long-term elevations in air pollution levels (such as those present in many major cities) have been shown to be associated with increased mortality. This increase most probably is attributable to elevated mortality in individuals already at high risk, such as the elderly and the infirm. Numerous studies of the health effects of long-term elevated exposures have revealed associations with chronic respiratory disease in adults (both prevalence and exacerbation of symptoms), and with changes in pulmonary function and acute lower respiratory tract illnesses in both adults and children (National Academy of Sciences 1979; American Thoracic Society/American Lung Assoc. 1978; Ferris 1978; Holland et al. 1979; Shy 1979).

Animal and epidemiological studies (McJilton et al. 1973; Amdur and Corn 1963; Amdur 1971; USEPA 1974) suggest that oxidation products of SO_2, namely sulfuric acid and particulate sulfates, possibly acting synergistically with SO_2 and other pollutants such as nitrates, particulates, and ozone, are the primary causative agents. SO_2 smoke and suspended particulates and, more recently, suspended sulfates have been used as indices of pollution and there is as yet no firm evidence as to which substance or substances are the causative agents.

Experiments exposing volunteers to sulfuric acid have had various results. Observed effects include transient cough, bronchoconstriction, increased pulmonary resistance, changes in specific airway conductance, and alteration of bronchial mucociliary clearance at exposure concentrations ranging from 100 $\mu g/m^3$ to 39,000 $\mu g/m^3$ (Morrow et al. 1980; Sim and Pattle 1957; Lippmann et al. 1980). On the other hand, in a number of studies, no adverse effects were reported at levels up to 1000 $\mu g/m^3$ (Sackner et al. 1977; Sackner et al. 1978; Newhouse et al. 1978).

The National Air Surveillance Network (NASN) has collected data on sulfate levels in the United States. NASN estimated an annual average global background level of 1-2 $\mu g/m^3$ (USEPA 1975). There is a 24-state region in the northeastern U.S. that has consistently higher levels of pollutants than the rest of the country. In 1972, urban air in that area had an annual average sulfate level of 13.6 $\mu g/m^3$, with 10.2 $\mu g/m^3$ for nonurban air. The annual average of urban air in the rest of the country was 7.9 $\mu g/m^3$, with 4.4 $\mu g/m^3$ for nonurban air.

Human health effects of sulfates have been reported in experimental and epidemiological studies. The major epidemiological studies have been done by the Community Health and Environment Surveillance System (CHESS) of the Environmental Protection Agency. In one CHESS study, a 10%

increase in the rate of asthma attacks was found when the minimum temperature was above 50°F and the level of suspended sulfates (SS) was greater than 10 $\mu g/m^3$. The threshold level was estimated to be 12 $\mu g/m^3$ of SS when the minimum temperature was between 70° and 50°F (Finklea et al. 1974a). Another CHESS report estimated the threshold level for effects to be 1.4 $\mu g/m^3$ at temperatures greater than 50°F (Finklea et al. 1974b).

Cohen et al. (1972) studied asthmatics in West Virginia who lived near a coal-fired powerplant. Although no single pollutant could be determined to be the prime cause of asthma attacks, "...suspended sulfate levels showed the strongest association with attack rate after the effects of temperature were removed." A sulfate level of 20 $\mu g/m^3$ was used as the concentration demarcating days of high and low pollution levels.

The best judgment of the threshold for effects of SS on cardiopulmonary symptoms in the elderly was estimated to be 6 $\mu g/m^3$ (24-hr average) (Stebbings and Hayes 1976). An increase in chronic bronchitis was noted in adults exposed to four to seven years of 90-95 $\mu g/m^3$ of SO_2, 15 $\mu g/m^3$ of SS, and 60 $\mu g/m^3$ of TSP. In another study, an increase in acute lower respiratory disease was found in children exposed to three or more years of 177 $\mu g/m^3$ of SO_2, 65 $\mu g/m^3$ of TSP, and 7 $\mu g/m^3$ of SS (USEPA 1974).

Sulfate levels of 9-15 $\mu g/m^3$ were estimated to be the thresholds for increased acute respiratory disease and chronic bronchitis. Concentrations of 6-10 $\mu g/m^3$ of SS were estimated to aggravate asthma (USEPA 1975; Colucci 1976).

Both SO_2 and TSP are referred to as criteria pollutants because NAAQS exist for them. However, nonattainment, especially in urban areas, is not uncommon. At the present time there are no ambient air quality standards for SO_2 transformation products such as sulfuric acid and sulfates, or for the respirable fraction of the TSP that serves to transport volatile materials to sites deep within the lung.

2.3 PHOTOCHEMICAL OXIDANTS, NITROGEN OXIDES, AND HYDROCARBONS

Photochemical oxidants are formed in the atmosphere from nitrogen oxides and hydrocarbons. Ozone is the largest component, but compounds such as peroxyacetyl nitrate (PAN), acrolein, peroxybenzoyl nitrates (PB_2N) and aldehydes probably are responsible for the acute irritative effects on the eyes, nose, and throat associated with photochemical air pollution. Efforts to identify excess mortality associated with increased levels of photochemical air pollution have not been successful.

On the basis of studies by Renzetti and Gobran (1957), Richardson and Middleton (1958), and Hammer et al. (1974), there is evidence of nose, throat, and eye irritation in the range of 200-294 $\mu g/m^3$. Cough and chest discomfort increased markedly when a level of 588 $\mu g/m^3$ was reached. Motley et al. (1959) reported decreased pulmonary function among patients with chronic respiratory disease exposed to unfiltered Los Angeles air relative to filtered air. This effect was reversible. The levels of exposure were estimated to be in the range of 200-294 $\mu g/m^3$. Bates and Hazucha (1973) observed a reduction in pulmonary function among healthy subjects at an ozone level of 725 $\mu g/m^3$, and an enhancement of this effect in the presence of SO_2 at 969 $\mu g/m^3$.

The principal man-made sources of nitrogen oxides are coal, oil, natural gas, and motor vehicle fuel combustion. High levels of NO_2 (above 282 $\mu g/m^3$, or 150 ppm) can be lethal (National Research Council 1977; Shy et al. 1973). Nonurban regions average 8 μ/m^3 (4 ppb) NO_2 and 2 $\mu g/m^3$ (2 ppb) NO, whereas in urban areas, concentrations of nitrogen oxides are 10-100 times higher. Ten percent of the cities in the United States with populations less than 50,000 and 54% of those with populations between 50,000 and 500,000 have a yearly average nitrogen dioxide concentration equal to or exceeding 0.06 ppm, the lower limit at which health effects were noted in a community study in Chattanooga (Shy et al. 1970a, 1970b); 85% of the cities in the 7,500,000 population class exceed this yearly average. Meeting the current Ambient Air Quality Standard for nitrogen oxides (0.05 ppm or 100 $\mu g/m^3$ expressed as the yearly arithmetic mean concentration) would be more difficult in the large population concentration in the Northeast.

Laboratory and epidemiologic studies indicate slight increases in respiratory symptoms and illness, slight or no difference in pulmonary function, and little or no change at all in healthy subjects (Ferris 1978). Information on chronic nitrogen dioxide poisoning is extremely scarce because (1) no response is observed until a critical concentration is reached, (2) damage develops slowly, and (3) nitrogen dioxide is usually associated with other pollutants. According to the intensity and duration of exposure, respiratory illness ranges from slight irritation to burning and pain in the throat and chest to violent coughing and shortness of breath (Waldbott 1973).

Hydrocarbons enter the air from both natural and man-made sources, with fossil-fuel combustion contributing only a very small amount of the total. Generally, concentrations of the primary hydrocarbons released into the air are too low to cause any observable health effects.

2.4 RESPIRABLE PARTICULATES

The U.S. Environmental Protection Agency (USEPA) was charged with completing a review of the Clean Air Act and promulgating revised standards by December 1980. The role of the fine particulates in the development of human respiratory disease has elicited much response from both the public and scientific community during USEPA's period of comment solicitation. As a result of public discussion and research activities, a number of definitions and parameters have evolved that will likely be incorporated into any revised particulate standard (Miller et al. 1979).

Particulates derived from fossil-fuel combustion may be crystalline or amorphous forms, fibers, spheres, or aggregate meshes (Holland et al. 1979) on which transformation products, toxic elements, and organic molecules can adsorb. Characteristics of physical deposition in biological systems support a size discrimination between "fine" and "coarse" at approximately 2 μ (Lippman 1977, Lippmann et al. 1979; Palmes and Lippman 1979; Altshuler et al. 1957, 1967). At diameters less than 2.5 μ, total alveolar deposition ranges between 15 and 25%. Maximal deposition of approximately 45% occurs during mouth breathing. Since alveoli are nonciliated and comprise the gas-exchange surface of the deep lung, any material that is preferentially deposited in these structures requires careful scrutiny. This is particularly pertinent for particles less than 0.5 μ in linear diameter, which can undergo systemic transport in the body via diffusion. As a result of these findings, USEPA's Health Effects and Environmental Science Research Labs are recommending to the Office of Air Quality Planning and Standards a cutoff of 2.5 μ aerodynamic for setting a size-specific standard.

It is to be expected that fine particles are preferentially deposited in the alveolar region, where the deposition fraction can equal 65% (Task Group on Lung Dynamics 1966). Although the relative importance of removal mechanisms in the alveoli presently is unclear, there is substantial agreement that deposition in these tissues has damage potential.

A number of toxic elements released to the atmosphere during coal combustion for power generation are found preferentially associated with small particle emissions. It is thought that these elements and/or their compounds volatilize during combustion and condense or adsorb onto the fine particles that are not captured by existing precipators or flue-gas desulfurizers (Natusch et al. 1979). Those elements in airborne fly ash that exhibit the most pronounced trends of increasing concentration with decreasing diameter are lead, thallium, antimony, cadmium, selenium, arsenic, nickel, chromium, and zinc (Davison et al. 1974). Similar inverse relationships between fly ash particle size and element concentration have been noted by Lee and von Lehmden (1973) and Toca (1972). Other elements found in fly ash include iron, manganese, vanadium, silicon, magnesium, carbon, beryllium, and aluminum; these exhibit only limited concentration trends (Davison et al. 1974).

Some compounds found concentrated on the surfaces of fine particles are not only known to be toxic, but are also suspected carcinogens. Particulate polycyclic organic matter (POM) and compounds of the elements lead, cadmium, selenium, arsenic, nickel, and chromium fall into this latter category (Natusch 1978). High concentrations of arsenic, nickel, cadmium, and particularly sulfur are found in a 300-angstrom-thick shell on fly ash surfaces (Natusch 1978). This ready availability of toxic elements and large surface area provided by fine particles allows intimate contact with body fluids and alveolar tissues (Natusch 1978). As a result, legitimate concern exists that the manifest health effects of exposure may alter all bodily systems rather than respiratory alone (U.S. Office of Technology Assessment 1979).

Approximately 50% of total sulfur released is associated with the smallest size fraction. Most of this atmospheric sulfur is thought to be in the form of sulfuric acid mists or metallic sulfate compounds. Some health analysts now consider these transformation products to be at least partly responsible for the inhalation hazard presented by gaseous sulfur oxides (Natusch 1978). Other co-contaminants include nitrogen dioxide and nitrate and hydrocarbon aerosols.

Studies of fine particulate exposure in healthy and asthmatic individuals have been performed with inert dusts and powders such as $CaCO_3$. Functional changes, i.e., airway flow resistance, gas trapping, and abnormal intrapulmonary mixing have been observed after brief inhalation of particles ≤ 1 μ in diameter (Dubois and Dautrebande 1958; Lovejoy et al. 1961). These changes may be caused by edema, excessive mucus secretion, or constriction of smooth muscle, trachea, or bronchii (Amdur and Underhill 1968). The exact mechanism is unknown. The additional alteration of impaired gas exchange also has been noted (Lovejoy et al. 1961).

Epidemiologic evaluations have been confined largely to major, acute episodes in which large numbers of people were stricken. The Donora, New York City, Meuse Valley, and London episodes are notable. At the time of these incidents, both investigators and instrumentation were insensitive to the potential role played by respirable particles in mortality and morbidity induction. Perhaps the data collected during the recent 10-year monitoring program initiated by the Harvard School of Public Health in six communities throughout the United States will clarify the potential linkages between fine particles and human health (Ember 1977). The best available evidence indicates that inhalation exposure to fine particles is likely to be harmful.

2.5 TRACE ELEMENTS

The amount of trace elements emitted from coal combustion may be highly variable, based on (1) the type of coal used, (2) the engineering of the powerplant, and (3) the analytical methods used to determine the elements. The amount and relative percentages of the trace elements incorporated in coal depend on the geology of the coal-forming basin (Zubovic 1975).

During combustion, trace elements tend variably to concentrate in three fractions that ultimately effect their emission: Group I--those that are not volatilized, but melt and become bottom ash and slag or are emitted from the stack as fly ash (barium, chromium, cerium, cobalt, europium, hafnium, lanthanum, manganese, rubidium, scandium, samarium, strontium, tantalum, and thorium); Group II--those that are volatilized but condense out on the fly ash particles as the flue gas cools (arsenic, beryllium, cadmium, copper, gallium, molybdenum, nickel, lead, antimony, selenium, uranium, vanadium, and zinc); and Group III--those that are volatilized and remain almost completely in the gas phase (mercury, chlorine, and fluorine) (Klein et al. 1975; Ray and Parker 1977).

Trace elements in outdoor air are associated primarily with particulate matter (Friedlander 1973). Natusch et al. (1979) found that arsenic, antimony, cadmium, lead, selenium, and thorium were most concentrated on the smallest respirable particules due, in theory, to preferential adsorption. These particles are the most difficult to control using pollutant removal equipment. For reasons of toxicity, bioaccumulation, and widespread exposure, trace elements of potential health concern from increased coal utilization include lead, mercury, arsenic (AsIII), uranium, selenium, nickel, cadmium, fluorine, beryllium, chromium (Cr IV), antimony, bromine, copper, gallium, iodine, iridium, molybdenum, zinc, thallium, tin, titanium, uranium, radon, and thorium (Comar and Nelson 1975; Van Hook 1979; Morrow et al. 1977; U.S. Office of Technology Assessment 1979; Heit 1977; Santhanam et al. 1979). A panel of toxicologists (Morrow et al. 1977) compared predicted concentrations of certain trace elements with derived acceptable concentrations and concluded there is highly suggestive evidence that beryllium, nickel, cadmium, chromium (Cr IV), arsenic (As III), fluorine, and uranium may present the greatest potential problems among the trace elements listed above. Van Hook (1979) adds mercury to this list and the U.S. Office of Technology Assessment (1979) adds selenium. The "Rall Report" (U.S. Department of Health, Education and Welfare 1978) also notes the potential additional health effect of trace elements acting as catalysts for converting SO_2 to acid sulfates. However, trace element emissions are expected to assume a minor role in comparison to other pollutants emitted by coal combustion and in comparison to other sources of these trace elements.

Consideration of the cellular effects of the trace elements is complex. Physicochemical factors such as oxidation states and covalent bonding of the trace element ion affect its biological efficacy. Conjoint exposure to nonmetal toxicants can affect cellular response. Finally, many of the metals exhibiting deleterious effects are nutritionally required, in some cases at a level not much below that at which toxic effects are evident.

2.6 RADIONUCLIDES

Coal contains small quantities of U-238, U-235, Th-232 and their radioactive daughter products. The results of a survey of the uranium and thorium concentrations in coal samples from all regions of the United States demonstrate that the radionuclide content of coal is quite variable both between and within the various coal supply regions (see Table 4.20). As coal is burned, some of this natural radioactivity is released into the atmosphere as fly ash; the remainder is in the bottom ash, and its fate is dependent upon ash disposal practices.

Exposure to radioactivity in fly ash is mainly by inhalation of small particles, ingestion of contaminated terrestrial foods, and direct radiation from deposition on ground surfaces. The ingestion pathway represents the largest potential exposure mode, but this pathway is highly dependent upon the quantity of agricultural crops growing in the environs of a coal-fired plant. Therefore, coal-fired plants in urban areas with little agriculture could be expected to have less exposure via ingestion of contaminated food. The dose due to inhalation would be the major pathway of exposure. However, more people may live in the urban setting, so that population dose (man-rem) could be as high as when ingestion pathways are significant. In any case, population doses are comparable to those for persons exposed to radiation from routine releases from nuclear powerplants. Moreover, the combustion of coal containing more than 1 ppm uranium and 2 ppm thorium, or fly ash treatment systems that release more than 1% of the ash, could be expected to lead to higher radiation doses than those cited here.

In any case, the radiation doses from coal-fired plants (as cited in McBride et al. [1978]) are far below those incurred from naturally occurring background radiation and thus are not expected to result in any increased risk to public health.

2.7 ORGANIC MATTER

Polynuclear organic matter (POM) may be formed during the combustion or pyrolysis of fossil fuels or other materials containing carbon and hydrogen. The two classes of POM compounds found most often in ambient air are polycyclic aromatic hydrocarbons (PAHs) and their neutral nitrogen analogues (Santodonato et al. 1979). These two chemical groups have been shown to contain a number of carcinogenic agents (National Academy of Sciences 1972). As a consequence, the PAHs, and more specifically the potent carcinogen benzo(a)pyrene (BaP), have been the most extensively studied POM compounds.

At the present time, BaP concentrations in the atmosphere provide the most widely available indicator of PAHs. While some investigators have found good correlations between BaP and other PaHs (Sawicki 1967), this relationship is likely to depend on and vary with the type of POM emission sources (National Academy of Sciences 1972; Santodonato et al. 1979).

Some data exist on the magnitude of POM exposures from coal-fired powerplants. Diehl et al. (1967) have determined the emission levels of three polynuclear hydrocarbons from coal-fired units of varying type and size. The National Academy of Sciences (1972) has estimated BaP emissions from coal-fired powerplants on a nationwide basis to be about 1 ton per year compared to a total of 1320 tons per year from all major source categories. The National Adademy of Sciences ratio of 1:1320 implies that total BaP emissions from coal-fired powerplants are neglible compared with emissions from other sources. However, older measurements using in-stack collection methods of fly ash sampling may reflect only a small fraction of the total organic material emitted (Natusch 1978; Van Hook and Shults 1976) because most organic emissions are in the vapor phase within stacks and do not condense onto the surfaces of fly ash particles until the stack plume has cooled to 100-200°F, some distance from the stack mouth.

The association between POM and particles in the atmosphere is important in the consideration of health effects. Many studies indicate that BaP is associated primarily with particulate matter, the great majority of which is in the respirable size range, about 0.1 to 2 μm in diameter Santodonato et al. 1979). (POM preferentially condenses onto small particles because of their larger surface-to-volume ratio.) Thus, almost all the POM emitted from coal-fired powerplants is capable of respiratory tract deposition, given the joint presence of small particles upon which it may be absorbed.

An additional important factor in determining POM concentrations is the efficiency (or completeness) of combustion. The concentrations of BaP associated with coal combustion can vary by a factor of 10,000, depending on the efficiency of the system in question; the BaP emission factor for efficient, modern utility plants is 20-400 $\mu g/10^6$ Btu, while the corresponding figure for hand-stoked residential coal furnaces is 1,700,000-3,300,000 $\mu g/10^6$ Btu (National Academy of Sciences 1972). Some sources (U.S. Department of Health, Education and Welfare 1978; Spengler et al. 1979) have indicated concern over the possibility of increased POM emissions from smaller, less efficient, coal-fired industrial facilities located in or near high-density population areas. "While modern utility plants are designed to efficiently utilize the fuel value of the coal (and thereby reduce POM emissions), the conversion of older industrial facilities, from oil and gas to coal would expose a large population to elevated concentrations of POM. This is particularly significant for those older facilities with low stacks located in urban areas" (Spengler et al. 1979).

The atmospheric stability of POM and its ability to be transported from the emission source to other locations is pertinent to the assessment of possible population exposures. Evidence suggests that many POM compounds oxidize or photodegrade at a significant rate under atmospheric conditions. The atmospheric lifetime of a POM particle is related to particle size, with small (<1 μm in diameter) particles having a longer atmospheric residence time. However, the reaction rates of compounds on smaller particles is likely to be faster because of the greater surface area exposed to oxidants and light (Santodonato et al. 1979). In contrast, there is some evidence that many POM compounds are relatively stable in the atmosphere in the form in which they are naturally present and are able to travel distances of several hundred miles (Lunde and Bjorseth 1977).

A number of POM compounds have been detected in ambient air, but the most extensive monitoring data exist for BaP. Urban BaP concentrations in the U.S. have been declining steadily with average concentrations of 6 ng/m^3 in 1958 (Sawicki 1967), 3.2 ng/m^3 in 1966, and 0.5 ng/m^3 in 1975 (Santodonato et al. 1979). The decline is attributed primarily to decreases in residential coal combustion and restrictions on open burning.

Exposure to relatively high levels of POM-containing substances has resulted in various non-neoplastic skin and eye responses in a number of clinical and occupational settings. Skin effects have been produced in the clinical setting through the application of coal tar and coal solutions to the skin of human subjects, often in conjunction with exposure to ultraviolet light. Responses to this type of exposure include tar phototoxicity or erythema (Tanenbaum et al. 1975; Kaidbey and Kligman 1977), decreased mitotic activity (Fisher and Maibach 1973),

and induction of cutaneous aryl hydrocarbon hydroxylase, a carcinogen-metabolizing enzyme that may play a role in the induction of cancer (Bickers and Kappas 1978).

Non-neoplastic skin effects also have been observed in occupational settings. These include nonallergic and allergic dermatitis, phototoxicity and photoallergic reactions, folliculitis, acne, and pigment disturbances. Effects have occurred following exposure to coal tar and coal tar products, pitch, creosote, asphalt, and petroleum products (National Academy of Sciences 1972). Additional sources have reported the occurrence of acute eye effects following exposure to coal tar pitch, including inflammation, conjunctivitis, and reduction in visual acuity (Emmett et al. 1977).

Skin carcinomas historically have been observed in occupations having exposure to high-temperature coal tar products (SRI International 1977; National Academy of Sciences 1972; Henry 1947). Epidemiological studies of occupational groups exposed to POM, many among coke plant workers (Lloyd 1971) and gas production workers (Doll et al. 1965) and others from the roofing and asphalt industries (Hammond et al. 1976), have shown that long-term exposure to the products of coal distillation is associated with an elevated rate of lung cancer and, in some cases, cancer of other sites.

Community air pollution studies typically involve much lower (two to four orders of magnitude) levels of ambient exposure than the levels observed in occupational settings. In these studies an investigation is made of the possible associations between community mortality and morbidity rates and some direct or indirect measure of air pollution.

In a considerable number of studies, urban and rural populations have been compared in an attempt to attribute any differences in rate to greater air pollution in urban areas. These studies tend to show, in males, a fairly consistent excess (about two-fold) in urban lung cancer rates over those found in the corresponding rural areas (Manos and Fisher 1959; Levin et al. 1960; Haenzel et al. 1962; Stocks and Campbell 1955). However, at present there is no general consensus that these differences are due to air pollution. Community study efforts also have correlated cancer mortality rates with indices of air pollution, but the results of these studies have been inconsistent.

The number and complexity of chemical structures of materials derived from coal combustion can produce a wide variety of cellular effects. Some types of POM are toxic to various types of cells upon acute exposure. Others are not toxic until they are acted upon by intracellular enzyme systems, which convert them into various metabolites, some of which can be toxic. Many of these compounds also are mutagenic when tested in appropriate mutagenesis assay systems, although some of the compounds must again be enzymatically altered in order to induce mutagenesis. Some of these POM (or their metabolites) also can cause certain morphological and biochemical alterations in cell cultures exposed to them, and some of these altered cells can eventually become tumorigenic when injected into the appropriate host animal (a process known as transformation). The major concern of much of this research is the potential carcinogenicity of at least some of the coal-associated chemicals.

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3. CRITERIA POLLUTANTS AND THEIR TRANSFORMATION PRODUCTS

The three major types of air pollution from fossil fuel-combustion sources are as follows. (1) The sulfur oxide and particulate complex generated by the combustion of sulfur-containing fossil fuels, especially coal and crude oil. The sulfur oxide component includes sulfur dioxide (SO_2), sulfuric acid, and sulfate aerosols. The particulate component consists of soot, fly ash, metallic oxides, and suspended sulfates and nitrates. (2) Photochemical oxidants formed in the atmosphere from a chemical reaction between solar radiation and nitrogen oxide and hydrocarbons. Combustion in coal and oil-fired powerplants produces small amounts of hydrocarbons relative to motor vehicle emissions, but contributes relatively sizable amounts of nitrogen oxides. (3) Trace emissions of heavy metals such as lead, cadmium, arsenic, and mercury. Residues of such heavy metals tend to accumulate in the body over time.

The major categories of health effects associated with air pollution are (1) development or aggravation of chronic respiratory disease; (2) exacerbation of asthma and allergic conditions; (3) development of acute and chronic respiratory disease in children; (4) aggravated cardio-pulmonary disease; (5) air pollution-associated mortality; (6) development of symptoms such as cough, mucous membrane irritation, and fatigue; and (7) breathing impairment. The relationships between exposures to specific agents and these health effects are summarized in the following sections. This type of information, along with the information in Table 3.1, which provides a summary of emissions by sources, is useful in assessing cost-effective pollution control strategies and the relative health risks attributable to coal combustion versus other pollution sources.

3.1 THE SULFUR OXIDE/PARTICULATE COMPLEX

3.1.1 Introduction

These two pollutants are discussed together because they tend to originate from a common source--combustion of sulfur-containing fossil fuels--and hence are present together in the atmosphere. The sulfur oxide/particulate complex is the major component of air pollution generated by coal-fired powerplants. As shown in Table 3.1, these sources account for 62% of all emissions of sulfur oxides and for 22% of all particulate emissions from electric utilities in the U.S.

Sulfur oxides in the atmosphere occur in three principal forms: sulfur dioxide, sulfuric acid, and inorganic sulfates. The Continuous Air Monitoring Project, which monitors six large U.S. cities, and the National Air Surveillance Network (NASN), which collects 24-hour sampling data for about 100 locations throughout the country 26 times per year, have produced a large body of data on ambient levels of sulfur dioxide. As shown in Figure 3.1, annual average concentrations measured by the NASN show a declining trend. Sulfuric acid and inorganic sulfates are not routinely measured. NASN estimated an annual average global sulfate background level of 1-2 $\mu g/m^3$ (USEPA 1975). There is a 24-state region east of the Mississippi (roughly bounded by Illinois and Massachusetts to the north and Tennessee and North Carolina to the south) that has consistently higher levels of pollutants than the rest of the country. In 1972, urban air in that area had an annual average level of 13.6 $\mu g/m^3$ for sulfates, and nonurban air 10.2 $\mu g/m^3$. The annual average for urban air in the rest of the country was 7.9 $\mu g/m^3$, with 4.4 $\mu g/m^3$ for nonurban air.

About 98% of the sulfur released into the air is in the form of sulfur dioxide (SO_2), and most, if not all, of the remainder is sulfuric acid (Rall 1974; Goldstein 1975). Sulfur dioxide and sulfate in the atmosphere are derived from both natural and anthropogenic sources. Hydrogen sulfide, which comes mainly from the action of anaerobic bacteria, is oxidized to form sulfur dioxide (Grey and Jensen 1972 as cited in Goldstein 1975). Sulfur dioxide is a colorless gas having a pungent irritating odor, which is detectable at concentrations of about 0.5 to 1 ppm (2860 $\mu g/m^3$) (U.S. Dept. of Health, Education and Welfare 1970a). SO_2, naturally occurring and formed by the combustion of fossil fuels, is oxidized to form sulfate. The three principal sulfate compounds formed by the oxidation of SO_2 are sulfuric acid, ammonium sulfate, and ammonium bisulfate (Frank 1978). The formation of H_2SO_4 by the oxidation of SO_2 to SO_3 and the subsequent reaction of SO_3 with water occurs spontaneously in clean air and sunlight at a rate of 0.1%/hr (Goldstein 1975). Sulfuric acid also is formed by photochemical reactions in the presence of hydrocarbons, nitrogen oxides, and activated forms of oxygen.

Table 3.1. Estimated Emissions of Air Pollutants
(percentage of national total in 1975)

Source	Sulfur Oxides	Total Suspended Particulates (TSP)	Nitrogen Oxides	Carbon Monoxide	Hydrocarbons
Coal electric utilities	62	22	29		
Coal industrial combustion	7	8	8		
Oil electric utilities	6		4		
Oil industrial combustion	2		3		
Transportation	2	7	51	76	60
Industrial processes (iron, steel, copper, smelters, oil refining, etc.)	13	8		17	14
Resident-commercial heating	4	2	3		
Structural material		37			
Cement, sewage, asphalt		8			
Coal transportation		2			
Other	4	6	2	7	26

A blank indicates zero or a negligible (<1) number.

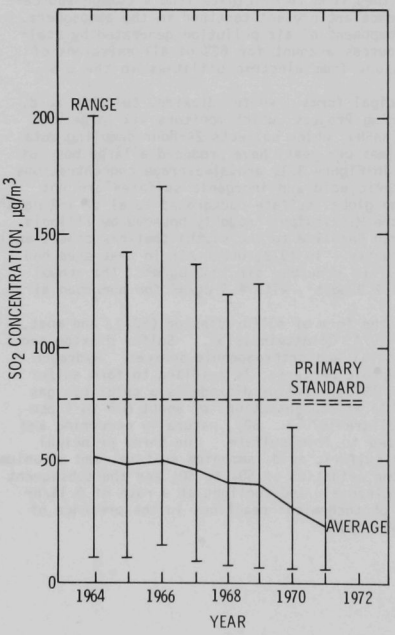


Fig. 3.1.

Average Sulfur Dioxide (SO_2) Concentrations for 32 Urban NASN Stations. From National Research Council (1975).

Sulfuric acid and other sulfates typically account for about 5 to 29% of the total suspended particulate matter found in urban air (U.S. Dept. of Health, Education, and Welfare 1970a). The size of the sulfate particle is important, since the location of deposition in the respiratory system depends on the size of the particle and the velocity of air movement (American Thoracic Society, American Lung Assoc. 1978). Larger particles are deposited in the nasopharynx, while smaller particles (mass median diameter of 1 μm or less) are deposited in the tracheobronchial tree and alveoli. Studies of the particle-size distribution of suspended atmospheric sulfate show that 80% or more of urban atmospheric sulfate is associated with particles less than 2 μ in diameter, and thus is largely in the respirable size range. According to Brosset (1973), the majority of airborne sulfates are less than 0.5 μm in diameter.

Particle-size distribution is a significant factor not only in the determination of health effects, but also in the comparison of the various techniques used in measuring particulates. The high-volume (HV) sampling method typically used in the U.S. measures total suspended particulates collected on filters, and yields values that are considerably greater than the corresponding values obtained by the British smoke filter method (BS), which is largely the basis for British data on atmospheric particulate concentrations.

Historically, the association between sulfur oxide/particulate air pollution and mortality has been demonstrated by acute air pollution episodes in such places as the Meuse Valley, Belgium, in 1930; in Donora, Pennsylvania, in 1948; and in London, in 1952 (U.S. Dept. of Health, Education and Welfare 1970a). Excess deaths recorded during these three episodes were 63, 20, and 4000 respectively. In addition, these episodes caused a marked increase in the incidence of respiratory and cardiovascular illness. As a consequence of these air pollution disasters and their apparent association with ambient levels of SO_2, there was a major emphasis on SO_2 in the setting of air quality standards under the United States Clean Air Act Amendments passed in 1970. However, subsequent toxicologic studies (Amdur 1957; Amdur 1959; Amdur and Underhill 1968; and Alaire et al. 1972) indicated that SO_2 by itself could not be the primary causative agent. Instead, a combination of SO_2 and particulates was postulated as the cause. Other animal and epidemiologic studies (McJilton et al. 1973; Amdur and Corn 1963; Amdur 1971; and USEPA 1974) suggest that oxidation products of SO_2, namely sulfuric acid and particulate sulfates, possibly acting synergistically with SO_2 and other pollutants such as nitrates, particulates, and ozone, are the primary cause of the adverse health effects associated with SO_2. Because there is no conclusive evidence as to which substance or substances are the true causative agents, SO_2, smoke, and suspended particulates, and more recently suspended sulfates, have been used as indices for sulfur oxide air pollution. More information is needed to develop the most efficient pollution control strategy for sulfur oxides. In particular, although the CHESS studies reawakened the concern about sulfates, the CHESS data need further evaluation. A qualitative and quantitative summary of the health effects of sulfur oxides and particulates follows.

3.1.2 Summary of Health Effects

Sulfur dioxide (SO_2), one of the earliest suspected toxic agents in air pollution episodes, is a potent pulmonary irritant at concentrations exceeding 10 to 20 ppm. Such concentrations are virtually never encountered in ambient air, however. A few sensitive individuals may show minor changes in lung function at concentrations of 1 to 2 ppm, but long-term animal exposures at these levels reveal no apparent detrimental effects. These findings are supported by studies on laboratory animals and human volunteers (Berry et al. 1974; Rall 1974). The apparent reason for the low potency of SO_2 is that SO_2 is nearly completely absorbed in the mucous linings of the upper airways and does not reach the more sensitive regions of the lungs. Sulfur oxides penetrate into the lungs only when they are adsorbed onto the surface of particulate matter (5 μ-diam or less) or aerosols such as fog, and hence are in the form of sulfuric acid or sulfates. These products of SO_2, respirable particles and aerosol, are more highly irritating than SO_2 (Rall 1974). In addition, they can act synergistically with SO_2, increasing its toxicity in the upper respiratory system. The animal studies on these combined effects and their conclusions are discussed in Section 3.2.2.1 (below), and human laboratory studies in Section 3.2.2.2.

3.1.2.1 Animal Studies

Effects of simultaneous exposure to SO_2 and irritant gas on guinea pigs was studied by Amdur et al. (1975). Aerosols of soluble salts of ferrous iron, manganese, and vanadium produced an enhancing effect on the toxicity of SO_2, although the concentrations used (0.7-1.0 $\mu g/m^3$) were considerably greater than any levels of metals reported in urban air. Frank et al. (1964) examined the response of human subjects to levels of SO_2 of about 1, 5, and 15 ppm, with or without the addition of sodium chloride aerosol. No constant enhancing (potentiation) effect was demonstrated. In animal studies, three- to four-fold potentiation of the irritant response to SO_2 was observed in the presence of particulate matter capable of oxidizing SO_2 to sulfuric acid (U.S. Dept. of Health, Education and Welfare 1970a). The degree of potentiation was related to the concentration of particulate matter. Sulfuric acid, in combination with particulate sulfates, also produced bronchoconstriction in guinea pigs. Response was highly dependent on particle size, and the smallest particles showed the greatest irritant potency. In both guinea pigs and man, sulfuric acid and irritant particles have a greater irritant potency than SO_2 alone (U.S. Dept. of Health, Education and Welfare 1970a).

The following series of human laboratory studies with sulfur oxides highlight the difficulty of establishing dose-response relationships in health research on air pollution.

3.1.2.2 Human Laboratory Studies

Experiments exposing volunteers to sulfuric acid have had varying results. Amdur et al. (1958 as cited in Sackner et al. 1978) exposed volunteers to 5-10 minutes of 0.35-5.0 mg/m^3 of H_2SO_4. At concentrations less than 1 mg/m^3, sulfuric acid could not be detected by odor, taste, or irritation. H_2SO_4 could be detected by all subjects at concentrations greater than 3 mg/m^3. There was coughing when the subjects inhaled deeply at 5 mg/m^3. Morrow et al. (1980 as cited in Lippmann 1980) found small changes in specific airway conductance in normal volunteers exposed to 1000 mg/m^3 of H_2SO_4 for 10 to 60 minutes.

Sackner and coworkers (Sackner 1976; Sackner and Reinhardt 1977; Sackner et al. 1977) exposed human subjects and stable asthmatic children to aerosols of sulfates and sulfuric acid mist. Particle sizes were 1 µm or less. Exposures to sulfuric acid were in concentrations of 10, 100, and 1000 µg/m^3. Sulfates were in similar particle sizes and concentrations, and included ammonium sulfate, zinc sulfate, and sodium sulfate. Exposures lasted 10 minutes. Extensive tests of pulmonary function before and after exposure did not reveal any consistent adverse effects at any of the exposure levels. In addition, no significant changes in health status were reported 3 months after exposure when the subjects and patients were restudied. The asthmatic children were not exercised during the exposure. The sulfuric acid and types of sulfates used certainly are appropriate to represent ambient exposures, as are the sulfate levels (up to 1 mg/m^3) and the particle sizes. Newhouse et al. (1978) studied the effects of H_2SO_4 at the industrial Threshold Limit Value (TLV) of 1 mg/m^3. They found a speeding of bronchial clearance in healthy volunteers who exercised while being exposed to sulfuric acid.

Lippmann et al. (1980) exposed ten healthy nonsmokers to H_2SO_4 at concentrations of 100, 300, and 1000 µg/m^3 for one hour. There were no significant changes in respiratory mechanics; however, bronchial mucociliary clearance was altered. Based on both human and animal studies, ". . . it appears that chronic H_2SO_4 exposures at concentrations of ~100 µg/m^3 could produce persistent changes in mucociliary clearance in previously healthy individuals and exacerbate preexisting respiratory disease" (Lippmann 1980).

Several extensive reviews of the epidemiology of sulfur oxide air pollution are available in the literature (World Health Organization 1972; National Research Council 1978; Ferris 1978; Holland et al. 1979; Shy 1979). Because of the limitations of each type of research used in investigating environmental health, there is apparent inconsistency between the toxicological and the epidemiological literature on sulfur oxides. The levels at which epidemiological studies have shown evidence of excess mortality from sulfur oxide exposure are well below the levels suggested by toxicological studies. Frank (1968) tried to explain this phenomenon with the argument that "the constituents of air pollution are many, are complex, and are not completely known; they have potential for additive, cumulative and synergistic effects; they may interact with age, smoking, climate, ill health, etc. By contrast, the variables in a toxicological experiment are kept few and regulated." Furthermore, the levels at which several epidemiological studies have shown evidence of excess mortality are questionable because of the nature of the data and methodology used (Mazumdar and Redmond 1980). Relevant results are summarized below under the categories of short-term and long-term health effects.

Short-Term Health Effects

Numerous epidemiological studies have demonstrated an association between short-term acute exposure to the sulfur oxide-particulate complex and increased risk of illness and death. In particular, increases in mortality during the acute air pollution episodes such as occurred in London, Meuse Valley, and Donora have been recorded. Table 3.2 contains estimated numbers of deaths attributable to high concentrations of air pollution, as calculated from the difference between the number of excess deaths recorded during the episode and the number expected for that same time period. Also in Table 3.2, estimated excess deaths are related to measurements of SO_2 and particulate matter or smoke, where these data are available.

Excess deaths were attributed primarily to bronchitis, pneumonia, and cardiac diseases, and were found particularly among persons aged 45 years or older. These and other episodes drew the attention of the medical community to the public health dangers of uncontrolled air pollution. Reduced levels of air pollution resulting from antipollution measures since 1962 have made it difficult to detect changes in mortality using subsequent data. The maximum concentration of smoke reported during the 1952 fog in London which caused 4000 deaths, 4460 µg/m^3, is more than 100 times the current average values seen in the U.S., where particulates exceed 200 µg/m^3 (HV) as an annual mean, or where the individual 24-hour values are much above 500 µg/m^3 (HV).

Table 3.2. Selected Air Pollution Episodes

Year	Days	Place	Estimated Excess Deaths	Maximum 24-Hour Pollution ($\mu g/m^3$) Smoke	SO_2
1930	December	Meuse Valley, Belgium	63	--	--
1948	October	Donora, Pennsylvania	20	--	--
1948	November 26-December 1	London, England	750	2780	2150
1952	December 5-8	London, England	4000	4460	3830
1956	January 3-6	London, England	1000	2830	1430
1957	December 2-5	London, England	750	2417	3335
1959	January 26-31	London, England	250	1723	1850
1962	December 3-7	London, England	700	3144	3834
1963	November	New York City	2000	--	--

From Holland et al. (1979).

In studies of day-to-day variations in mortality in London (Martin and Bradley 1960; Lawther 1963) during the period 1952-1962, significant increases in deaths (defined as 20 or more deaths of a total of 200-400 per day) were considered to be associated with 24-hour average concentrations of smoke of more than 750 $\mu g/m^3$ with SO_2 levels of about 710 $\mu g/m^3$. Studies of minor episodes in New York (occurring in 1953 and 1962) by Greenberg et al. (1962, 1963) showed that increases in excess deaths were evident when 24-hour average smoke levels were equivalent to about 570-720 $\mu g/m^3$ (BS) with SO_2 concentrations between 850 and 1750 $\mu g/m^3$. The London and New York data, the most extensive and useful data on short-term effects, are reasonably consistent in that increased deaths were discernible between 1952 and 1967 when smoke levels exceeded 500-800 $\mu g/m^3$ (BS), along with SO_2 concentrations exceeding approximately 700-1000 $\mu g/m^3$. Based on this and similar evidence, the World Health Organization adopted the figure of 500 $\mu g/m^3$ for each pollutant as the minimum associated with short-term changes in mortality in 1972. It has not been as readily possible to detect variations in daily deaths at lower concentrations due to confounding factors such as temperature extremes, season, and influenza epidemics. For instance, for two recent episodes in London reported in the winter of 1975-76, it was not clear whether the small changes in mortality observed were related directly to pollution or to the associated cold weather. During one of these episodes (December 15-16, 1975), smoke and SO_2 concentrations exceeded 500 $\mu g/m^3$ and 1000 $\mu g/m^3$, respectively, the highest levels recorded in London in ten years.

High SO_2 and TSP pollution often is associated with low temperatures, and the relationship between low temperatures and increased mortality among the elderly has been established by Russell (1926). The presence of cofactors such as temperature contributes to the disagreement about the magnitude of short-term health effects of air pollution. Future studies on short-term effects must involve data associated with sharp temperature changes, but fairly constant pollution levels, in order to identify the true causes of adverse health. In view of the possible trade-off between the health benefits of pollution control and those of reducing the cost of heating or cooling homes, it is important to assess the relative importance of temperature versus pollution on public health. According to MacFarlane (1976), any short-term effects on mortality arising from smoke concentrations below 1000 $\mu g/m^3$ (24-hour average, BS) and 1000 $\mu g/m^3$ of SO_2 are not in any way outstanding in comparison with those of sharp changes in temperature.

Other studies of short-term health effects (Buechley et al. 1973; Schimmel and Murawski 1976; Schimmel 1978; Wyzga 1978; Mazumdar et al. 1980) cast doubt on the effects of SO_2 per se on mortality and indicate a greater association between daily mortality and particulates. In particular, using the New York-New Jersey data for the period 1962-1966, Buechley et al. (1973)

noted that mortality was 2% greater than expected on days with SO_2 levels above 500 $\mu g/m^3$. However, extending the data base to 1971-1972, they continued to find slightly higher mortality on high-SO_2 days even though the absolute amounts of SO_2 were one-tenth of what they were in 1967-1968. Buechley et al. thus concluded that SO_2 was acting as a surrogate for some other substances that had not changed significantly.

In several studies conducted in Britain and in the United States an attempt has been made to relate day-to-day variation in morbidity to SO_2, particulates, and suspended sulfates. These have been reviewed by Ferris (1978) and Holland et al. (1979). In the 1960s increased illness was discernible among bronchitic patients when 24-hour average concentrations of smoke (BS) exceeded 250 $\mu g/m^3$ (TSP[HV] equivalent of about 350 $\mu g/m^3$) with SO_2 concentrations of 500 $\mu g/m^3$ (Lawther et al. 1970). Again, however, it is difficult to separate effects on morbidity at lower pollution levels from those of adverse meteorological conditions. Among the U.S. studies, those conducted by USEPA's Community Health and Environmental Surveillance System (CHESS) are the most comprehensive, although their methodology has come under considerable criticism. In the CHESS studies (USEPA 1974), health indicators used for short-term effects were aggravation of cardiopulmonary symptoms and asthma. Threshold limits corresponding to worst-case, least-case, and best-judgment estimates were developed for the effects of SO_2, suspended particulates, and suspended sulfates from the data obtained. A summary of these limits is provided in Table 3.3.

One CHESS study in the New York area (Finklea et al. 1974a as cited in Goldstein 1975) found no consistent simple correlation between the attack rate of asthma and individual pollutants. When the minimum temperature was between 30 and 50°F, there was estimated to be a 13% increase in risk when levels of total suspended particulates (TSP) were 76-260 $\mu g/m^3$. There was a 9% increase in risk at 8.1-10.0 $\mu g/m^3$ of suspended sulfates and an 8% increase when the 24-hour concentration was greater than 10 $\mu g/m^3$. When the minimum temperature was above 50°F and the suspended sulfate level was greater than 10 $\mu g/m^3$, there was a 10% increase in attack rate. When the minimum temperature was below 30°F, the threshold estimate of suspended sulfates was 7.3 $\mu g/m^3$. The threshold level was estimated to be 12 $\mu g/m^3$ when the minimum temperature was between 30 and 50°F. Asthmatics in the Salt Lake Basin were studied also (Finklea et al. 1974b as cited in French 1975). The highest morbidity rates were found to be associated with high levels of suspended sulfate. At temperatures greater than 50°F, the threshold level for effects was estimated to be 1.4 $\mu g/m^3$.

The CHESS studies indicate that adverse effects on elderly subjects having heart and lung disease and on asthmatics were experienced on days when the 24-hour levels of SO_2 and TSP concentrations exceed the levels prescribed in the National Primary Standards. These adverse health effects, however, appear to be associated with suspended sulfates rather than SO_2 and TSP, as evidenced by the consistency of relationship between symptom aggravation and sulfate levels and the lack of consistency of this relationship with the other pollutants. These studies again underscore the relative innocuousness of SO_2 at current ambient levels and the need for further efforts to quantify the effects of particulates and sulfates.

Long-Term Health Effects

Long-term health effects of air pollution have been investigated by comparing mortality rates in areas of heavy pollution with those in relatively cleaner areas. The most difficult problem faced in these studies is identifying and adjusting for the various factors contributing to geographic differences in mortality. Important factors include cigarette smoking, occupation, socioeconomic status, age, pre-existing disease, and population migration. Many investigations have shown that cigarette smoking, in particular, has an overwhelming effect on morbidity and mortality, especially from cardiopulmonary disease, and that the etiologic contribution of air pollution is in fact relatively small. This is the main reason why many epidemiological studies that take into account cigarette smoking fail to detect adverse long-term health effects of air pollution. Goldstein (1975) estimates that 70% of chronic respiratory disease is attributable to cigarette smoking, while only 10% of the incidence of these diseases may be associated with SO_2/particulate exposure. That the effects of air pollution at present ambient levels are overshadowed by those of cigarette smoking is an important point to consider in assessing health effects from fuel combustion.

Tables 3.4 and 3.5 are summaries of several studies dealing with the association between residence in highly polluted areas and mortality or prevalence of chronic respiratory disease. Although intervening variables might explain some of the air pollution-health effect associations listed in Tables 3.4 and 3.5, the consistency of these associations cannot always be explained by these variables. In particular, in the studies of chronic bronchitis cited in Table 3.5, information was generally obtained on smoking, socioeconomic level, age, sex, and other confounding factors. The weight of evidence suggests that although cigarette smoking exerts an overall stronger adverse effect on life expectancy and disease rates, air pollution is at least additive to, and possibly acts synergistically with, cigarette smoking.

Table 3.3. Summary of Estimated Threshold Limits for Adverse Effects of Short-Term Exposure to Sulfur Oxides and Particulates

Adverse Effect	Type of Estimate	Minimum Temperature (°F)	Daily Average Levels Linked to Adverse Health Effects[a] ($\mu g/m^3$)		
			Sulfur Dioxide (SO_2) (365 $\mu g/m^3$)[b]	Total Suspended Particulates (TSP) (260 $\mu g/m^3$)[b]	Suspended Sulfates (SS) (no standard)[b]
Aggravation of cardio-pulmonary symptoms in elderly					
"Well" panel	Worst case	20-40	81-365	NPE	<1
	Least case		NE	NE	NE
	Best judgment		NPE	NPE	8-10
	Worst case	>40	81-365	68	2
	Least case		NE	NE	10
	Best judgment		NPE	80-100	8-10
"Heart" panel	Worst case	>40	NPE	76-260	10
	Least case		NE	NE	10-20
	Best judgment		NPE	NPE	10
"Lung" panel	Worst case	20-40	NPE	76-260	6
	Least case		NE	NE	NE
	Best judgment		NPE	NPE	10
	Worst case	>40	NPE	76-260	11
	Least case		NE	NE	12
	Best judgment		NPE	NPE	12
"Heart and lung" panel	Worst case	20-40	181	47	9
	Least case		NE	NE	10
	Best judgment		NPE	80-100	10
	Worst case	>40	NPE	76	6
	Least case		NE	NE	17
	Best judgment		NPE	NPE	10
Aggravation of asthma	Worst case	30-50	NPE	61-75	8
	Least case		NE	NE	NE
	Best judgment		NPE	105	9-10
	Worst case	>50	23	61-75	<1
	Least case		NE	NE	10
	Best judgment		180-250[c]	70	8

[a] NE - no effect below Primary Standard, or simply no effect for suspended sulfates, for which no Primary Standard has been established. NPE - no proven effect below Primary Standard, for simply no proven effect for suspended sulfates.

[b] National Primary Air Quality Standard.

[c] This judgment is based on presently summarized studies and on a previously reported CHESS study of asthma in New Cumberland, West Virginia.

Table 3.4. Association of Geographic Differences in Mortality with Long-Term Residence in Areas of High Air Pollution (as indicated by sulfur oxides and particulate levels)

Reference[a]	Characteristics of the Study	Findings
Pemberton and Goldberg (1954)	Bronchitis mortality rates for males 45 years and older in county boroughs of England and Wales, 1950-1952.	Sulfur oxide levels (sulfation rates) were consistently correlated with bronchitis death rates in the 35 county boroughs analyzed.
Buck and Brown (1964)	Bronchitis mortality rates in 214 areas of Britain, evaluated with respect to SO_2, particulates, social index, and population density, 1955-1959.	Social index and SO_2 accounted for 36% of the variation in bronchitis mortality within county and noncounty boroughs and in urban districts. Within London boroughs, social index was most important factor.
Zeidberg et al. (1967)	Deaths for each cause in Nashville, Tenn., categorized into three levels of air pollution and three levels of economic class, 1949-1960.	Within the middle social class, total respiratory disease mortality, but not bronchitis and emphysema mortality, was significantly associated with sulfation rates and social index. White infant mortality rates were significantly related to sulfation rates.
Winklestein et al. (1967, 1968)	One-hundred-twenty (120) census tracts in Buffalo stratified into four levels of particulate pollution and cross-stratified into five levels of economic status.	Within economic level, death rates of white males aged 50-69 years, for all causes and for chronic respiratory disease, corresponded to the gradient of particulate, but not SO_2, pollution.
Lave and Seskin (1970)	Multiple regression analysis of mortality as a function of air pollution, population density, socioeconomic level, age, and race in 114 Standard Metropolitan Statistical Areas in the United States, 1960.	Air pollution (particulates and sulfates) was a statistically significant variable in the regression models for total death rates and death rates for infants less than 1 year old.
Watanage and Kaneko (1971)	Mortality rates in Osaka, Japan, stratified by air pollution level, 1965-1966.	There was a stepwise increase in total mortality and deaths from circulatory disease in areas of greater pollution.

From Shy (1978).

[a]References cited are in the source, and are omitted from this report.

Table 3.5. Studies of Air Pollution (as indicated by sulfur oxide and particulate levels) and Prevalence of Chronic Respiratory Symptoms

Reference[a]	Characteristics of the Study	Findings
Ferris and Anderson (1962); Anderson and Ferris (1965); Anderson et al. (1965)	Questionnaire and ventilatory function survey of random sample of adult population in Berlin, N.H., and Chilliwack, British Columbia, 1961-1963.	No apparent excess in symptom prevalence in association with air pollution. Diminished pulmonary function in residents of more polluted community, although differences in occupation, climate, and ethnic factors may account for these findings.
Holland et al. (1965)	Questionnaire and ventilatory function survey of outdoor telephone workers aged 40-59 in London, rural England, and East and West Coasts of United States.	Increased prevalence of respiratory symptoms, adjusted for smoking and age, a larger volume of morning sputum, and a lower average ventilatory function in London workers, and in the English compared with the American workers.
Petrilli et al. (1966)	Study of respiratory symptoms in nonsmoking women aged 65 and over who had not worked in industry and were long-time residents of their neighborhoods, 1961-1962.	Strong association between chronic respiratory symptom prevalence and area gradient for particulates and SO_2. Comparability between residents of suburban and industrialized areas not assured by exclusion of persons on social welfare assistance.
Bates et al. (1966)	Comparison of symptom prevalence, work absence, and ventilatory function in Canadian veterans residing in four Canadian cities.	Lower prevalence of symptoms and work absences and better ventilatory function in veterans living in the least polluted city.
Lambert and Reid (1970)	Postal survey of nearly 10,000 British residents, aged 35-64, using standardized questionnaire.	Among smokers, prevalence of chronic respiratory symptoms increased with increasing air pollution. No apparent symptom-pollution association in nonsmokers.
Ferris et al. (1973)	Repeat survey of residents of Berlin, N.H., six years after the original 1961 study.	Slightly lower prevalence of chronic respiratory symptoms and improved ventilatory function tests after standardization for age, sex, and smoking habits. Results attributed to decrease in air pollution levels.
Tsunetoshi et al. (1971)	Prevalence survey (Medical Research Council questionnaire) of residents of nine areas of Osaka and Hyogo prefectures, Japan, aged 40 and over.	Multiple regression analysis revealed increasing prevalence of chronic bronchitis, adjusted for age, sex, and smoking, corresponding to the area gradient of air pollution.

From Shy (1978).

[a]References cited are in the source, and are omitted from this report.

Cohen et al. (1972) studied asthmatics in West Virginia who lived near a coal-fired powerplant. Although no single pollutant could be determined to be the prime cause of asthma attacks, ". . . suspended sulfate levels showed the strongest association with attack rate after the effects of temperature were removed." A sulfate level of 20 $\mu g/m^3$ was used as the concentration demarcating days of high and low pollution levels.

Chapman et al. (1974 as cited in Goldstein 1975) reported on a CHESS study of children in Birmingham, Alabama, and Charlotte, North Carolina. They found that the forced expiratory volume of children in Birmingham (TSP = 127 $\mu g/m^3$ and SS = 13.3 $\mu g/m^3$) was less than that of children in Charlotte (TSP = 70 $\mu g/m^3$ and SS = 9.7 $\mu g/m^3$).

Shy et al. (1973) measured forced expiratory volume in children in New York. The older children had been exposed to 364-436 $\mu g/m^3$ of SO_2, 156-200 $\mu g/m^3$ of TSP, and 18-28 $\mu g/m^3$ of suspended sulfates the first five to ten years of their lives. Although there were no real differences in ventilatory function among younger children, ventilatory functions of older children from neighborhoods of high pollution were below those of children from neighborhoods of low pollution.

Stebbings and Hayes (1976) studied the effect of air pollution on cardiopulmonary symptoms in the elderly in New York. Air pollution had the strongest effect on the well elderly and nearly every pollutant, including suspended sulfate, had an effect. The best judgment of the threshold for effects of sulfate was estimated to be 6 $\mu g/m^3$ (24-hour average).

Chapman et al. (1973) reported on a Chicago study of more than 40,000 military inductees. The frequency of chronic bronchitis in whites was significantly greater in areas of high pollution (SO_2 = 90.5 $\mu g/m^3$, TSP = 129 $\mu g/m^3$, and SS = 14.5 $\mu g/m^3$).

The effects of air pollution on schoolchildren and adults in the Salt Lake Basin was reported in a Utah CHESS study (as cited in Knelson 1978). There was an increase in chronic bronchitis in adults exposed to 4-7 years of 90-95 $\mu g/m^3$ of SO_2, 15 $\mu g/m^3$ of SS, and 60 $\mu g/m^3$ of TSP. The prevalence of lower respiratory disease increased in children who had lived two or more years in neighborhoods of high pollution. In another CHESS study (as cited in Knelson 1978), adverse health effects were associated with pollution levels in smelter communities in Idaho and Montana. Chronic bronchitis increased in adults exposed to levels of 200-400 $\mu g/m^3$ of SO_2 and 7-20 $\mu g/m^3$ of suspended sulfates (SS). Acute lower respiratory disease increased in children exposed to three or more years of 177 $\mu g/m^3$ of SO_2, 7 $\mu g/m^3$ of SS, and 65 $\mu g/m^3$ of TSP.

In a CHESS study in New York (as cited in Knelson 1978), an association was found between respiratory disease and high levels of atmospheric pollutants. There was a significant increase in the rate of chronic bronchitis among adults exposed up to twenty years of 140-400 $\mu g/m^3$ of SO_2 and 10-25 $\mu g/m^3$ of SS. Both adults and children showed an increase in frequency and severity of acute lower respiratory illness after being exposed to 2-3 years of 255-320 $\mu g/m^3$ of SO_2, 95-125 $\mu g/m^3$ of TSP, and 10-15 $\mu g/m^3$ of SS (annual average).

Quantification of the long-term health effects of air pollution is a difficult task and multiple regression analyses such as those done by Lave and Seskin (1977) and Winklestein et al. (1968) suffer from major flaws such as extrapolating individual pollutant exposure for residents of large metropolitan areas from single pollution monitors. However, as identified by Holland (1979), studies by Ferris et al. (1971, 1973, 1976) of a random sample of respondents in Berlin, New Hampshire, revealed a lack of health effect from TSP (HV) values in the range of 80-130 $\mu g/m^3$ (annual mean). Ferris et al. noted an improvement in respiratory symptoms and pulmonary function in the same persons surveyed in 1961 and 1967, as a result of a decline in annual TSP levels from 180 $\mu g/m^3$ (HV) to 131 $\mu g/m^3$ (HV). A follow-up study in 1973 showed that a further decline in particulates to 80 $\mu g/m^3$ annual average (even while SO_2 levels increased) was not accompanied by a change in pulmonary function or respiratory status. A similar conclusion was reached by Holland and Stone (1965), who compared respiratory symptom prevalence in men aged 40-49 years in San Francisco with that in Los Angeles for the years 1960 and 1962. The mean TSP levels in San Francisco were 70, 72, and 74 $\mu g/m^3$, while the corresponding figures in Los Angeles were 156, 162, and 163 $\mu g/m^3$. There was no evidence of lower disease incidence in San Francisco; in fact, chest illness was significantly more common in San Francisco.

Perspective studies of the type conducted by Ferris et al. (1971, 1973, 1976) dealing with morbidity present the best hope for establishing safe levels for air pollution. A relative risk assessment involving a comparison of impacts of variables such as temperature, socioeconomic status, and smoking with that of pollution on mortality and morbidity also is necessary to provide the proper perspective. In particular, cross-sectional data should be gathered and analyzed with this objective in mind. In any case, this approach is likely to be more fruitful than attempts to determine the absolute effects of pollution. After establishing the risk of pollution versus smoking, relatively reliable dose-response relationships currently available for cigarette smoking could be utilized to quantify the risk of pollution.

There are a number of studies that focus on the effects of air pollution on children. In nearly all reported studies, children residing in more polluted communities showed diminishing ventilatory function when compared with children of similar age, sex, race, and social class living in

less polluted areas. On the basis of a study by Lunne et al. (1970), Holland et al. (1979) set a particulate concentration of 240 $\mu g/m^3$ (HV) (in the presence of SO_2 levels of about 240 $\mu g/m^3$) as the threshold level below which there is no discernible health effect among children. On the other hand, Shy (1979) argues in favor of a threshold level lower than 200 $\mu g/m^3$. Again, there is a need to relate these impacts to those of other variables such as socioeconomic status.

A summary of CHESS studies relating long-term pollutant exposures to human health is given in Table 3.6. These data indicate that existing National Ambient Air Quality Standards for SO_2 and TSP in terms of annual averages are at least adequate. Laboratory studies on both humans and animals show little evidence of adverse effects of SO_2 alone at ambient levels. Animal and human laboratory data available over the range of ambient exposures are shown for SO_2 in Figure 3.2 and sulfates in Figure 3.3.

3.2 PHOTOCHEMICAL OXIDANTS, NITROGEN OXIDES, AND HYDROCARBONS

3.2.1 Photochemical Oxidants

3.2.1.1 Introduction

Substances in the ambient air that possess oxidizing properties greater than that of oxygen are termed "oxidants". In addition, because they are formed in the atmosphere from precursor pollutants (hydrocarbons and nitrogen oxides) with energy provided by solar radiation, these substances are termed "photochemical" oxidants. Ozone (O_3) is the largest component of the photochemical oxidant complex present in polluted atmosphere, and is used as an indicator of the amount of total oxidant present. Other active and irritating compounds such as peroxyacetyl nitrate (PAN), acrolein, peroxybenzoyl nitrates (PB_2N), and aldehydes usually are present in an oxidant mix but are not usually measured. However, they probably are responsible for the acute irritative effects on the eyes, nose, and throat associated with photochemical air pollution. Nitrogen oxides (covered separately because they are regulated separately due to their own toxic properties) also are important components of this complex.

3.2.1.2 Summary of Health Effects

The National Academy of Sciences (1977a) has reviewed the literature on the health effects of photochemical oxidants and has identified the problems in the evaluation of such effects. One important problem is that ozone (O_3) is not the only active component in photochemical pollution, and it may occur in conjunction with other regulated compounds.

The health effects of ozone are summarized in Tables 3.7 and 3.8. Efforts to identify excess mortality associated with increased levels of photochemical air pollution have not been successful. The general conclusion in these studies has been that the increased temperature associated with high oxidant levels has been the factor responsible for the excess mortality observed (Biersteker and Evendijk 1976; Massey et al. 1970; Oechsli and Buechley 1970). Shoettlin and Landau (1961) studied incidence of asthmatic attacks associated with photochemical air pollution, but did not find direct evidence of an effect. On the basis of studies by Renzetti and Gobran (1957), Richardson and Middleton (1958) and Hammer et al. (1974), there is evidence of nose, throat, and eye irritation in the range of 200-294 $\mu g/m^3$ ozone. Cough and chest discomfort increased markedly when a level of 588 $\mu g/m^3$ ozone was reached. Motley et al. (1959) reported decreased pulmonary function among patients with chronic respiratory disease exposed to unfiltered Los Angeles air relative to filtered air. This effect was reversible. The levels of exposure were estimated to be in the range of ozone of 200-294 $\mu g/m^3$. Wayne et al. (1967) observed that at levels of ozone above 200-294 $\mu g/m^3$, running times of long-distance runners were affected. In this group of epidemiologic studies, effects appear to develop in the range of 200-294 $\mu g/m^3$ (0.1-0.15 ppm) ozone. However, these levels were measured by the Potassium Iodide (KI) method, in which measurements of the actual levels of ozone tend to be overestimated. In view of the difference between the KI method and the USEPA-approved chemiluminescence method, the levels reported in earlier studies must be re-evaluated.

In the controlled human exposure studies summarized in Table 3.8, an attempt was made to identify the relative effects of the ozone component of photochemical air pollution. Bates and Hazucha (1973) observed a reduction in pulmonary function among healthy subjects at an O_3 level of 725 $\mu g/m^3$, and an enhancement of this effect in the presence of SO_2 at 969 $\mu g/m^3$. Even though other factors in photochemical oxidant pollution may cause irritations, ozone appears to produce symptoms in exposed healthy subjects at about 725 $\mu g/m^3$ and in hyper-reactive subjects studied in the laboratory at 490 $\mu g/m^3$ when these levels have been measured by chemiluminescence.

While chamber studies are useful in defining acute or short-term health effects, epidemiologic studies are useful for identifying effects of low-level chronic exposure. Linn et al. (1976) did not observe any differences in pulmonary function and chronic respiratory symptoms between urban office workers in Los Angeles and San Francisco. The oxidant levels involved were low:

Table 3.6. Summary of CHESS Studies Relating Long-Term Sulfur Oxide and Particulate Air Pollution Exposures to Adverse Effects on Human Health

Adverse Effect	Type of Estimate	Duration of Exposure (years)	Annual Average Levels Linked to Adverse Health Effects ($\mu g/m^3$)		
			Sulfur Dioxide (SO_2) (80 $\mu g/m^3$)[a]	Total Suspended Particulates (TSP) (75 $\mu g/m^3$)[a]	Suspended Sulfates (SS) (no standard)[a]
Increase in prevalence of chronic bronchitis in adults	Worst case	3	62	65	12
	Least case	10	374	179	20
	Best judgment	6	95	100	15
Increase in acute lower respiratory tract infections in children	Worst case	3	65	65	7.2
	Least case	3	177	102	15
	Best judgment	3	95	102	15
Increase in frequency or severity of acute respiratory illness in families	Worst case	1	50	104	14
	Least case	3	210	159	16
	Best judgment	3	106	151	15
Subtle decreases in childhood ventilatory function	Worst case	1	57	96	9
	Least case	9	435	200	28
	Best judgment	8-9	200	100	13

From USEPA (1974), Table 7.1.11, p. 7-16.

[a]National Primary Ambient Air Quality Standard; the particulate standard is a geometric mean; the equivalent arithmetic mean would be about 85 $\mu g/m^3$.

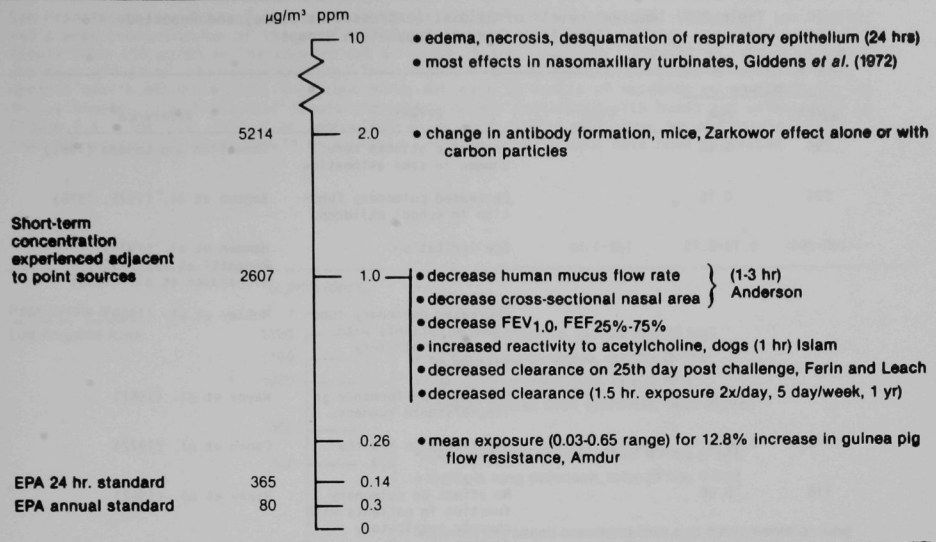


Fig. 3.2. Levels and Effects of Sulfur Dioxide (SO_2). From U.S. Office of Technology Assessment (1979).

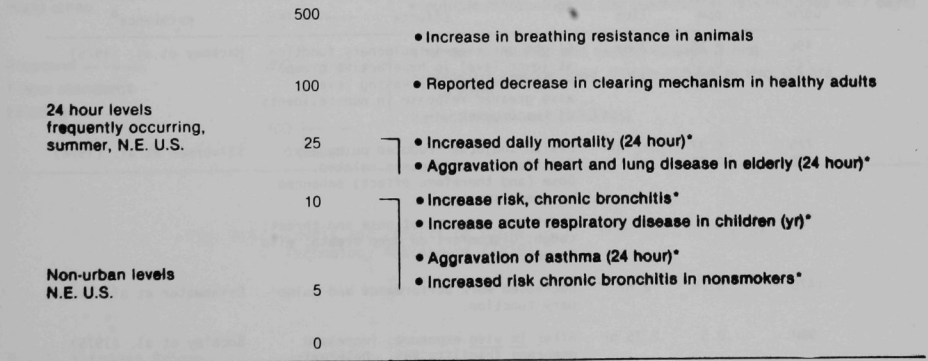


Fig. 3.3. Levels and Effects of Sulfates. From U.S. Office of Technology Assessment (1979).

Table 3.7. Ambient Levels of Oxidant (expressed as ozone) and Reported Effects on Various Population Groups

Exposure				
µg/m³	ppm	Time	Effects	Reference[a]
294	0.15		Asthmatic attacks more common in some asthmatics.	Shoettlin and Landau (1961)
294	0.15		Decreased pulmonary function in school children.	Kagawa et al. (1975, 1976)
196-294	0.10-0.15	1/2-1 hr	Eye irritation.	Hamman et al. (1974); Rensetti et al. (1957); Richardson et al. (1958)
			Decreased pulmonary function in patients with chronic respiratory disease.	Motley et al. (1959)
		1 hr	Decreased performance in long-distance runners.	Wayne et al. (1967)
118-216	0.06-0.11	Hourly values	No effects on healthy adults.	Cohen et al. (1972)
118	0.06		No effect on pulmonary function in patients with chronic respiratory disease.	Rokaw et al. (1962)

From Ferris (1978).

[a]References cited are in the source, and are omitted from this report.

Table 3.8. Ambient Levels of Ozone and Reported Effects on Human Beings--Selected Chamber Studies

Exposure				
µg/m³	ppm	Time	Effects	Reference[a]
490 725 980	0.25 0.37 0.50	2 hr	Slight decrease in pulmonary function at lower level in hyperactive group. Progresses with increasing levels; also greater response in non-residents of Los Angeles.	Hackney et al. (1975)
725 1470	0.37 0.75	1-2 hr	Health subjects--reduced pulmonary function; dose-response related. Dose (and therefore effect) enhanced by exercise.	Silverman et al. (1976)
			Symptoms: dryness of nose and throat; cough; discomfort on deep breath; mild nausea.	
1470	0.75	2 hr	Decreased work performance and pulmonary function.	Drinkwater et al. (1974)
980	0.5	2.75 hr	After in vivo exposure, increased membrane fragility RBS. Deleterious effects on cellular enzyme systems.	Buckley et al. (1975)
784-1176	0.4-0.6	1 hr	Increased airway resistance in healthy subjects.	Goldsmith et al. (1969)
294 588	0.15 0.30	1 hr	Decreased pulmonary function and increased respiratory symptoms. Effects enhanced by exercise at 65% maximum.	DeLucia et al. (1977)

From Ferris (1978).

[a]References cited are in the source, and are omitted from this report.

San Francisco had a mean ozone concentration of 39 $\mu g/m^3$ and a maximum of 549 $\mu g/m^3$; Los Angeles had a mean concentration of 137 $\mu g/m^3$, with a maximum of 1136 $\mu g/m^3$. Total suspended particulate levels were 135 $\mu g/m^3$ in Los Angeles and 47 $\mu g/m^3$ in San Francisco. Linn et al. noted an overwhelming effect of cigarette smoking. Thus, the current literature provides no evidence that chronic health effects result from continuous exposure to levels of oxidants currently occurring in Los Angeles. Environmental levels and standards are contrasted with human and animal data in Figure 3.4. The U.S. Office of Technology Assessment (1979) concluded, as did Ferris (1978), that the present standard should be observed until more valid data have been collected.

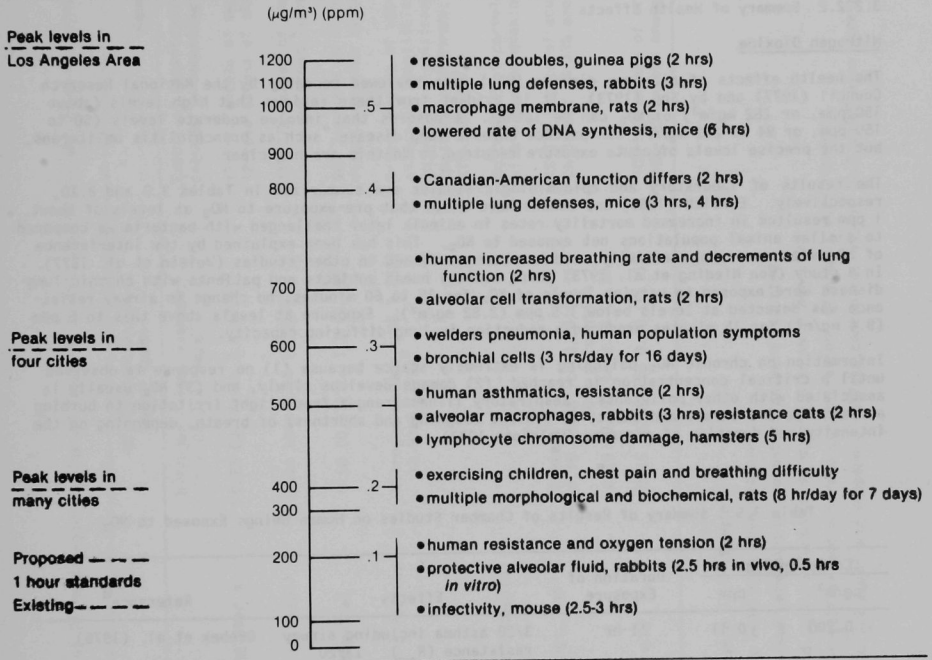


Fig. 3.4. Levels and Effects of Ozone (O_3). From U.S. Office of Technology Assessment (1979).

3.2.2 Nitrogen Oxides

3.2.2.1 Introduction

Nitrogen oxides are components of photochemical air pollution. Nitric oxide (NO) and nitrogen dioxide (NO_2) are formed from both natural and anthropogenic sources. The major natural source of NO is believed to be the anaerobic bacterial degradation of nitrogeneous compounds (Bowman 1977). The principal man-made sources of nitrogen oxides are coal, oil, natural gas, and motor vehicle fuel combustion. Coal combustion and transportation account for approximately 40 and 50% of the nitrogen oxide emissions. Coal-fired powerplants generally emit more nitrogen oxides

than do gas- and oil-fired plants because of the greater contribution to emissions by the nitrogen organically bound in the coal (Federal Interagency Committee 1980). Factories manufacturing explosives, fertilizers, and nitric acid, and metal-processing plants also are sources of NO and NO_2 (Bowman 1977). Nitric oxide eventually is converted to nitrogen dioxide, nitric acid, and nitrate aerosols by complex photochemical reactions (Brezonik 1978). NO and NO_2 are precursors of many different compounds--nitric acid, dinitrogen pentoxide, nitrates, peroxyacylnitrates, and N-nitrosamines.

Of the two oxides of nitrogen commonly monitored in ambient air, NO is relatively nontoxic at ambient concentrations, whereas NO_2 has effects on the respiratory system that are similar to those of O_3, but less severe. Atmospheric concentrations in nonurban regions average 8 $\mu g/m^3$ (4 ppb) NO_2 (1 ppm = 1880 $\mu g/m^3$), and 2 $\mu g/m^3$ (2 ppb) NO (1 ppm = 1230 $\mu g/m^3$) (USEPA 1971); in urban areas, concentrations of nitrogen oxides are 10 to 100 times higher. Daytime urban levels depend on motor traffic and sunlight, and peak concentrations rarely exceed 940 $\mu g/m^3$ (0.5 ppm).

3.2.2.2 Summary of Health Effects

Nitrogen Dioxide

The health effects of nitrogen dioxide (NO_2) were reviewed recently by the National Research Council (1977) and by Shy (1973). It is evident from these reviews that high levels (above 150 ppm, or 282 mg/m^3) of NO_2 can be lethal. Exposures that involve moderate levels (50 to 150 ppm, or 94 to 282 mg/m^3) can produce chronic lung disease, such as bronchiolitis obliterans, but the precise levels of acute exposure required to do this are not clear.

The results of laboratory and epidemiologic studies are summarized in Tables 3.9 and 3.10, respectively. Ehrlich (1966) and others have shown that pre-exposure to NO_2 at levels of about 1 ppm resulted in increased mortality rates in animals later challenged with bacteria as compared to similar animal populations not exposed to NO_2. This has been explained by the interference of NO_2 with macrophage activity, and has been confirmed in other studies (Voisin et al. 1977). In a study (Von Nieding et al. 1973) where healthy human subjects and patients with chronic lung disease were exposed to varying levels of NO_2 for 15 to 60 minutes, no change in airway resistance was detected at levels below 1.5 ppm (2.82 mg/m^3). Exposure at levels above this to 5 ppm (9.4 mg/m^3) for 15 minutes produced a reduction in lung-diffusing capacity.

Information on chronic NO_2 poisoning is extremely scarce because (1) no response is observed until a critical concentration is reached, (2) damage develops slowly, and (3) NO_2 usually is associated with other pollutants. Respiratory illness ranges from slight irritation to burning and pain in the throat and chest, to violent coughing and shortness of breath, depending on the intensity and duration of exposure (Waldbott 1973).

Table 3.9. Summary of Results of Chamber Studies on Human Beings Exposed to NO_2

Concentration		Duration of Exposure	Effects	Reference[a]
$\mu g/m^3$	ppm			
0.200	0.11	1 hr	3/20 asthma including airway resistance (R_{aw}). 13/20 increased sensitivity Carbacol.	Orehek et al. (1976)
0.564 (added to O_3 exposure)	0.3	2 hr	No increased response in "reactive" subjects.	Hackney et al. (1975)
1.316-1.880 (plus other components)	0.7-1.0	Not specified (until pulse 150/minute)	No significant change in reaction time or cardio-respiratory work efficiency in healthy subjects.	Holland et al. (1968)
<2.820	<1.5	0.25 hr	No change R_{aw} healthy subjects.	Von Nieding et al. (1973)

From Ferris (1978).

[a]References cited are in the source, and are omitted from this report.

Table 3.10. Summary of Epidemiologic Studies of Exposure to Nitrogen Oxides and Acute Respiratory Disease

Study Population	NO_x Concentration, mg/m^3 (ppm)	Reported Effect
Czechoslovakian children, aged 7-12 yr	Nitrogen oxides average = 0.02-0.07 mg/m^3[a]	Excess of hypertrophied tonsils and lymph nodes; changes in hematologic indexes.
USSR preschool and school children living near a fertilizer plant	0.32 (0.17) to 3.4 (1.8)[b]	17-fold excess of upper respiratory disease; 6- to 12-fold excess of abnormal chest films.
USSR adolescents in vocational training at chemical and fertilizer plants	Less than 0.10 (0.053)[c]	11%-27% excess of acute respiratory disease, increased blood lipoproteins, and chloesterol.
Residents living within 1 km of a USSR chemical works	0.58 (0.31) to 1.2 (0.64)[d]	44% increase in clinic visits for respiratory, visual, nervous system, and skin disorders.
Soviet children aged 8-11 yr living near a ferrous metallurgic plant	Nitrogen oxides = 46.3 (87) to 93.6 (176)[e]	5-fold excess of upper respiratory disease; 3-fold excess of tonsilitis; 2.5-fold excess of atrophic rhinitis; significant lag in growth, weight, and chest circumference; decreased urinary excretion of vitamin C.
Patients admitted to Philadelphia General Hospital for respiratory causes	--	No consistent correlation of respiratory admissions with nitrogen dioxide.
Chattanooga schoolchildren, their siblings, and parents	Average 0.15 (0.08) to 0.28 (0.15)[f] 90th percentile 0.19 (0.10) to 0.94 (0.50)	1%-17% excess of acute respiratory disease in children; 9%-33% excess in adults.
Chattanooga infants and children 6-9 yr old	0.15 (0.08) to 0.28 (0.15)[f]	10%-58% excess of acute bronchitis among infants; 39%-71% excess among 6- to 9-yr-old children.

From National Research Council (1977).

[a] Other pollutant exposres included SO_2 at 0.01-0.12 mg/m^3.

[b] High concentrations of SO_2 and sulfuric acid also were measured.

[c] Exposure to ammonia below the USSR maximum permissible concentration were reported.

[d] Concentrations of SO_2 (0.225 mg/m^3) and sulfuric acid (0.40 mg/m^3) also were measured.

[e] High concentrations of SO_2 and hydrogen sulfide and moderate concentrations of phenol were reported.

[f] Suspended nitrate concentrations of 3.8-7.2 $\mu g/m^3$ and suspended sulfate concentrations of 10.0 $\mu g/m^3$ also were measured.

The effects of community exposure to NO_2 were studied in four residential areas in Chattanooga, Tennessee. These studies linked high NO_2 exposure to an increase in childhood respiratory illness and a decreased respiratory performance of second-grade school children (Shy et al. 1970a, 1970b; Pearlman et al. 1971). In the first part of the Shy et al. study (1970a), one of the four areas in the Chattanooga study was in close proximity to a large TNT plant and had high NO_2 levels; another had high suspended particulate exposure; the remaining two areas served as controls. The mean range of daily NO_2 concentrations, measured over a six-month period, was between 117 and 205 $\mu g/m^3$, in areas of high NO_2 concentrations. The mean suspended nitrate level was 3.8 $\mu g/m^3$ or greater. The two control areas had average daily NO_2 levels of 118 and 80 $\mu g/m^3$ and suspended nitrate levels of 2.6 and 1.6 $\mu g/m^3$. The ventilatory performance of second-grade school children in the area of high nitrogen oxide exposure was significantly lower than the performance of children in the control areas. In the second part of the study (Shy et al. 1970b), a similar temporal pattern of respiratory illness was observed in the four areas during the 24-week study period. A consistent excess of respiratory illness was reported by families residing in the two exposed areas, particularly during the A_2/Hong Kong influenza epidemic and the period between the A_2 and influenza-B outbreaks. Differences in illness rates could not be explained by differences in family composition, economic level, or prevalence of chronic conditions. Parental smoking habits did not appear to influence respiratory illness rates of second-grade children. However, concentrations of suspended nitrates and TSP also were higher in the high-exposure groups as compared with the medium- and low-exposure communities. Suspended sulfates did not differ between communities, while concentrations of other possible contaminants such as SO_2 were not reported.

The National Air Surveillance Network (NASN) measured levels of NO_2 for the years 1967, 1968, and 1969 and found that yearly NO_2 averages reflect variations according to population densities. Since NO_2 does not exhibit marked seasonal variations, there was a direct comparison of the NASN yearly averages with the lower limit at which health effects were noted in Chattanooga studies (USEPA 1971). A concentration of 0.06 ppm (113 $\mu g/m^3$) or greater exceeds the Chattanooga health-effect-related NO_2 values. Ten percent of the cities in the United States having populations < 50,000 and 54% of those having populations between 50,000 and 500,000 have a yearly average NO_2 concentration equal to or exceeding 0.06 ppm; 85% of the > 500,000-population-class cities exceeded this yearly average. The current Ambient Air Quality Standard for nitrogen oxides is 0.05 ppm or 100 $\mu g/m^3$, expressed as the yearly arithmetic mean concentration. Morrow (1975) does not consider this figure conservative, after taking into account basic toxicological concepts and established procedures for setting safety standards for general populations.

All laboratory animals studied survived continuous exposures of a year or more to peak ambient concentrations of NO_2. These studies are summarized in Table 3.11; respiratory effects of chronic exposure are summarized in Table 3.12, and the pathology in animals exposed chronically are summarized in Table 3.13 (National Research Council 1977). The U.S. Office of Technology Assessment (1979) summarized the reported effects of low-level, short-duration exposure to NO_2, the levels reported in U.S. cities, and the proposed World Health Organization and USEPA standards; these are presented in Figure 3.5.

Other Oxides of Nitrogen

Dinitrogen pentoxide (N_2O_5), present in the atmosphere at very low concentrations, is formed by the following reactions (Colucci and Simmons 1978):

$$C_3 + NO_2 = NO_3 + O_2$$

$$NO_3 + NO_2 = N_2O_5$$

It is the unstable anhydride of nitric acid. In the presence of water vapor (e.g., high humidity), N_2O_5 can form nitric acid. This reaction takes place slowly in the gas phase, but more rapidly on a surface.

Nizhegorodov and Markhotskii (1971 as cited in Colucci and Simmons 1978) reported on workers exposed to low levels of CO, NH_3, and N_2O_5. They found relatively high levels of carboxy- and methemoglobin in the blood and excessive excretion of vitamin B_6 degradation products.

Peroxybenzoyl nitrate (PBzN) has not been found in ambient air in the United States, although it has been reported in the Netherlands (Meijer and Nieboer 1977 as cited in USEPA 1978). PBzN has been formed in a smog chamber by the reaction of benzaldehyde, ozone, and NO_2 (Heuss and Glasson 1968). Although there are no experimental or epidemiological studies on the health effects of PBzN, it is known to be extremely lacrimatory.

Peroxyacyl nitrates are believed to be formed by the photochemical reaction of acylperoxyl radicals with NO_2 (Haagen-Smit et al. 1953 as cited in Calvert and Thomas 1977). Peroxyacyl nitrates are highly unstable and have short half-lives. Peroxyacetyl nitrate (PAN) is the only member of the PAN family that has been identified in ambient air (Colucci and Simmons 1978).

Table 3.11. Survival of Animals Exposed Chronically to High Concentrations of NO_2

Animal	Concentration mg/m³	Concentration ppm	Duration of Exposure	Fatalities Attributed To Exposure
Mouse	0.94	0.5	12 months	None reported Pneumonitis
Rat	1.52	0.8	Lifetime	None
	3.76	2.0	Lifetime	None
	23.50	12.5	213 days	11% fatality
Guinea pig	7.52	4.0	4 hr/day, 5 days/week for 6 months	None
	28.20	15.0	6 months	
Squirrel monkey	1.88	1.0	16 months	None
Stump-tailed macaque	3.76	2.0	2 years	None
Dog	9.40	5.0	15 months	None
Rabbit	2.44	1.3	17 weeks	None

From National Research Council (1977).

Table 3.12. Respiratory Effects in Animals of Chronic Exposure to NO_2

Animal	Concentration mg/m³	Concentration ppm	Duration of Exposure	Effect
Rat	1.504	0.8	Lifetime	Tachypnea.
	3.760	2.0	Lifetime	Tachypnea, normal resistance, and dynamic compliance.
Guinea pig	9.4	5.0[a]	5.5 months	No change expiratory flow resistance.
Rabbit	1.88	1.0	18 months	Normal oxygen consumption.
	9.4	5.0	18 months	
	15.04-22.56	8.0-12.0	3-4 months	Decreased arterial blood oxygen static lung compliance, increase in nonelastic resistance and functional residual capacity. These effects disappeared when rabbits were permitted to breathe room air.
Dog	0.94-1.88	0.5-1.0[b]	16 hr/day for 18 months	Normal D_L CO.
Squirrel monkey	9.4	5.0	2 months	Tachypnea, decreased tidal volume, normal minute volume.
	9.4	5.0	2 weeks	Decreased tidal volume.
Stump-tailed macaque	3.76	2.0	2 years	Tachypnea.

From National Research Council (1977).

[a] For 4 or 7.5 hr/day, 5 days/week.

[b] Plus 0.250 mg/m³ (0.2 ppm) NO.

Table 3.13. Pathology in Animals Exposed Chronically to High Concentrations of NO_2

Animal	Concentration		Duration of Exposure	Pathology Attributed to Exposure
	mg/m^3	ppm		
Mouse	0.9	0.5	3 months	Ciliary loss, alveolar cell disruption
	0.9	0.5	6.18, and 24 hr/day for 3-4 mo.	Expanded alveoli, reduction of distal airway size, progressive parenchymal damage
	0.6-0.9	0.3-0.5	6 months	Destruction of bronchial epithelium, lymphocytic infiltration
	75.2	40.0	6-8 weeks	Epithelial abnormalities of terminal bronchioles
Rat	1.5-3.8	0.8-2.0	Lifetime	Ciliary loss, epithelial hyperplasia
	3.8	2.0	2 or more years	Thickened basement membrane
	31.9	17.0	20 months	Massive increase in collagen fibrils
	18.8-47.0	10.0-25.0	3 or more months	Enlarged thoracic cavities, dorsal kyphosis, distended alveoli and alveolar ducts
Guinea pig	9.4	5.0	7.5 hr/day, 5 day/week, 5.5 months months	Perivascular and tracheal inflammation, desquamative pneumonitis
	18.8	10.0	6 weeks	Hyperplasia of type 2 pneumatocytes
	28.2-37.6	15.0-20.0	2 hr/day, 5 day/week, 21 months	Inflammation of bronchiolar epithemlium
	41.4	22.0	2 hr/day, 3 weeks	Multifocal emphysematous changes
Rabbit	15.0-22.6	8.0-12.0	3-4 months	Necrosis of aveolar walls with enlargement of air spaces
	28.2-47.0	15.0-25.0	2 hr/day up to 2 years	No emphysematous lesions
Hamster	84.6-103.4	45.0-55.0	21-23 hr/day	Dilated alveolar spaces, inflammatory cells, epithelial hyperplasia, increased lung volume
Dog	9.4	5.0	15-18 months	No abnormalities
	48.9	26.0	6 months	Bullous emphysema
Squirrel monkey	9.4	5.0	169 days	Focal alveolar edema

From National Research Council (1977).

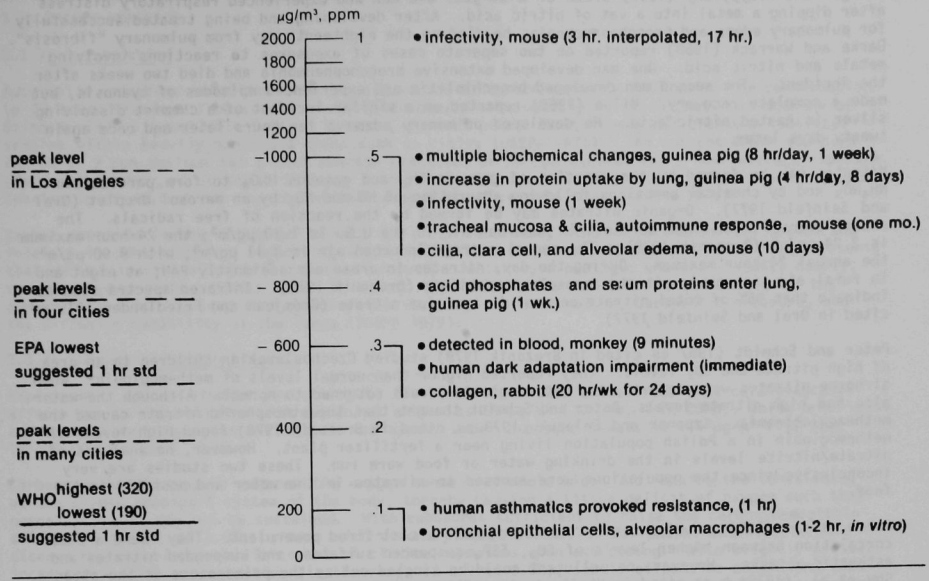


Fig. 3.5. Levels and Effects of Nitrogen Dioxide (NO_2). From U.S. Office of Technology Assessment (1979).

Smith (1965 as cited in Brezonik 1978) studied the effects of 1485 μg/m³ of PAN on cardiopulmonary functions of 32 college men. Respiratory rate, heart rate, and exhalation volume were not affected. There was increased O_2 uptake during exercise and recovery, but not at rest. Gliner et al. (1975 as cited in Colucci and Simmons 1978) found no health effects from exposure to 1188 μg/m³ of PAN. However, they reported that ". . . the subjects exposed to atmosphere containing PAN apparently suffered more subjective physical discomfort than subjects who did not receive PAN exposure."

There was no change in the maximal aerobic power of young men exposed to 1337 μg/m³ PAN for 16 minutes (Raven et al. 1974 as cited in Drinkwater et al. 1974). Drinkwater et al. (1974) reported no physiological effects to young men exposed to 1337 μg/m³ of PAN. In all cases, the subjects were exposed to PAN by breathing through a mouthpiece, since it is extremely lacrimatory.

N-nitrosamines have been reported around factories producing dimethyl and diethyl amines and in ambient air in New York City, Baltimore, Maryland, and Charleston, West Virginia (Colucci and Simmons 1978; Shapley 1976). They are believed to be formed by the reaction of secondary amines with nitrogen oxides or nitrous acid (Colucci and Simmons 1978). N-nitrosamines are known to be carcinogenic in animals, but there are no studies on the effects of atmospheric NNA on humans.

Nitric acid. The two major sources of nitric acid in the air are the reaction of NO_2 and a hydroxyl group and the reaction of N_2O_5 and water (Calvert and Thomas 1977). On a typical sunny summer day, the first reaction has a 50% conversion time of 2 to 3 hours (Davis et al. 1974 as cited in Spengler et al. 1975). The second reaction is less important.

Nitric acid vapor has a threshold limit value (TLV) of 5 μg/m³ for industrial exposure, since it is known to be an irritant (Brezonik 1978). There is little information on the effects of only nitric acid exposure, since most occupational exposures to nitric acid also involve NO_2 and possibly other chemicals. Schmid (1974 as cited in Colucci and Simmons 1978) reported that a 25-year-old truck driver who was exposed to fumes while cleaning up a spill involving 60% HNO_3 developed acute dyspnea three weeks after the incident and died four days later.

Treiger and Przypyszny (1947) wrote of a 58-year-old man who experienced respiratory distress after dipping a metal into a vat of nitric acid. After developing and being treated successfully for pulmonary edema and bronchopneumonia, he died on the eighteenth day from pulmonary "fibrosis". Darke and Warrack (1958) reported on two separate cases of exposures to reactions involving metals and nitric acid. One man developed extensive bronchopneumonia and died two weeks after the incident. The second man developed bronchiolitis and experienced episodes of cyanosis, but made a complete recovery. Milne (1969) reported on a similar incident of a chemist dissolving silver in heated nitric acid. He developed pulmonary edema a few hours later and once again twenty days later.

Nitrates may be formed by the reaction of gaseous NH_3 and gaseous HNO_3 to form particulate NH_4NO_3 and by chemical reactions following absorption of NO and NO_2 by an aerosol droplet (Orel and Seinfeld 1977). Organic nitrates may be formed by the reaction of free radicals. The annual average of nitrate aerosols in nonurban air in the U.S. is 1.10 $\mu g/m^3$; the 24-hour maximum is 3.24 $\mu g/m^3$ (Brezonik 1978). The annual average in urban air is 3.11 $\mu g/m^3$, with 8.90 $\mu g/m^3$ the annual 24-hour maximum. During the day, nitrates in urban air are mostly PAN; at night and in rural air, inorganic nitrate aerosols predominate (Brezonik 1978). Infrared spectra studies indicate that 95% of total nitrate aerosol is ammonium nitrate (Grosjean and Friedlander 1975 as cited in Orel and Seinfeld 1977).

Peter and Schmidt (1967 as cited in Brezonik 1978) studied Czechoslovakian children in an area of high nitrate and SO_2 levels. They reported higher-than-normal levels of methemoglobin. When airborne nitrates were removed, the methemoglobin levels returned to normal. Although the water also had high nitrate levels, Peter and Schmidt thought that the atmospheric nitrate caused the methemoglobinemia. Szponar and Belezuk (1973 as cited in Brezonik 1978) found high levels of methemoglobin in a Polish population living near a fertilizer plant. However, no analyses of nitrate/nitrite levels in the drinking water or food were run. These two studies are very inconclusive since the populations were exposed to nitrates in the water and possibly in the food.

Cohen et al. (1972) studied asthmatics living near a coal-fired powerplant. They found a clear correlation between higher levels of SO_2, TSP, suspended sulfates, and suspended nitrates and asthmatic attacks. However, no pollutant could be singled out as the prime cause of the attacks. Shy et al. (1970a,b as cited in Utell et al. 1979) studied Chattanooga schoolchildren and found a possible correlation between suspended nitrates and increased acute respiratory disease.

French et al. (1975 as cited in Colucci and Simmons 1978) reported on asthmatics in the New York-New Jersey metropolitan area in a USEPA CHESS study. They decided that nitrate levels of 8 $\mu g/m^3$ increased the rate of asthmatic attacks. Other reports from CHESS (as cited in Crocker et al. 1977) suggest that respiratory tract irritation may occur at nitrate levels of 3.8-7.2 $\mu g/m^3$.

Stebbings and Hayes (1976) conducted a USEPA CHESS study on the elderly in New York City. They reported that ". . . suspended nitrates may play a role in the exacerbation of pulmonary symptoms." A level of 2 $\mu g/m^3$ could be viewed as the "best judgment" estimate of the threshold for effects.

Utell et al. (1979) exposed 7 healthy volunteers and 11 asymptomatic asthmatics to 7000 $\mu g/m^3$ of $NaNO_3$ and 7000 $\mu g/m^3$ of NaCl (as a control) for 16 minutes. The mass median aerodynamic diameters (MMAD) of the $NaNO_3$ and NaCl particles were 0.49 $\mu g/m^3$, respectively. There was no significant response in either group of subjects. It was suggested that, while the smaller particles (MMAD 0.5-1.0 μm) of an aerosol are important because of their deep lung penetration, perhaps the larger particles (MMAD = 0.2-0.8 μm) induce bronchoconstriction and cough. If that is the case, the $NaNO_3$ particles used by Utell et al. were too small.

3.2.3 Hydrocarbons

3.2.3.1 Introduction

The NAAQS for hydrocarbons refer to nonmethane gaseous hydrocarbons in the atmosphere that can interact with O_3 and nitrogen oxides to form the irritating components of photochemical oxidant air pollution. Hydrocarbons enter the atmosphere from natural as well as man-made emissions. Transportation accounts for 60% of man-made emissions, while fossil-fuel combustion contributes only a small fraction of the total amount.

3.2.3.2 Summary of Health Effects

There are several hydrocarbon compounds that may induce adverse health effects. However, quantitative data on individual hydrocarbons in terms of their health effects are scarce. Generally, concentrations of the primary hydrocarbons released into the air are much too low to cause any observable health effects. However, ambient concentrations of hydrocarbons in the morning strongly influence the oxidant concentrations attained in the afternoon under appropriate meteorological conditions. Air quality data indicate that nonmethane hydrocarbon concentrations of 200 $\mu g/m^3$ (0.3 ppm as carbon) between 6 and 9 a.m. can produce a maximal hourly average oxidant concentration of 200 $\mu g/m^3$ (0.1 ppm) 2 to 4 hours later (U.S. Dept. of Health, Education and Welfare 1970b). Hence, the standard for hydrocarbons is set at 160 $\mu g/m^3$ for the early morning hours.

3.3 CARBON MONOXIDE

3.3.1 Introduction

Automobile emissions constitute the largest source of carbon monoxide (CO) while the contribution to CO concentrations from coal- or oil-fired powerplants usually is small (National Academy of Sciences 1977b) (see Table 3.14). However, combustion of fossil fuels can create a localized problem within heavily populated areas such as cities (USEPA 1979). The current USEPA standard for CO is 9 ppm maximum for 8-hour average exposure, or 35 ppm maximum for 1-hour average exposure (National Academy of Sciences 1977b). Carbon monoxide health effects in mammals are due to inhalation of ambient air and the normal catabolism of pyrrole rings (USEPA 1979).

Carbon monoxide is one of the pollutants for which laboratory studies have been very useful and relevant in developing and supporting a standard. This is because it is one of the few pollutants that has at least one specific reaction production which is a biologic measure of dose, namely, the carboxyhemoglobin (COHb) level in blood. The final level of COHb is determined by the concentration of inspired CO, alveolar ventilation, red cell volume, barometric pressure, and the diffusive capability of the lungs (USEPA 1979).

3.3.2 Summary of Health Effects

Experimental animal studies pertaining to low-level CO effects demonstrate that cardiovascular effects can be demonstrated with exposures as low as 58 $\mu g/m^3$ (30 ppm; 4-7% COHb), while behavioral and central nervous sytem effects do not appear under exposure levels of 115 $\mu g/m^3$ (100 ppm; 12-20% COHb) (USEPA 1979).

Carbon monoxide exposure reduces the oxygen content of whole blood and impairs the functioning of the oxygen transport system of the body, thereby causing a tissue deficit of oxygen such that normal function may not be sustained. With exposures sufficient to raise the carboxyhemoglobin saturation to 2.5 to 3.0%, persons with coronary heart disease or peripheral arteriosclerotic disease may experience earlier onset of clinical symptoms upon exercising. With slightly greater increases in COHb concentration (in the range of 3.0 to 6.5%), subtle effects on central nervous system function can be detected in normal individuals. Present air quality standards for carbon monoxide are such that an increase in COHb saturation to 1.5% is permitted. Estimated health effects levels for CO exposure are summarized in Table 3.15. The TLV (50 ppm) is set to prevent systemic intoxication (Sivulka 1980; American Conference of Governmental Industrial Hygienists (ACGIH) 1976).

It has been suggested that the principal mechanism of CO toxicity is a blocking of the energy flow on the cellular level through the cytochrome system, not hypoxemia, despite the inhibitory effects that CO has on oxygen uptake and release (Sivulka 1980; Goldbaum 1977; Goldbaum et al. 1975, 1976; Ramirez et al. 1974). Secondary mechanisms involve hypoxia, a deficiency of oxygen (Winter and Miller 1976).

3.4 LEAD

3.4.1 Introduction

Lead (Pb) comes under the third category of air pollution referred to in Section 3.1, trace emissions of heavy metals, residues of which tend to accumulate in the body over time. Among the common trace elements, lead is the only one that has been selected as a criteria pollutant and consequently is discussed here. This summary is based largely on a recent document on air quality criteria for lead prepared by the USEPA (1977).

The California Air Resources Board has adopted an air quality standard for lead based on health protection considerations. The standard is 1.5 $\mu g/m^3$, averaged over 30 days. A national air quality standard of 1.5 $\mu g/m^3$ for lead has also been established by USEPA. Comments addressing the proposed standard and USEPA's response to these comments are summarized in a USEPA (1977) document. The relevant issues are reexamined in a more recent report (National Research Council 1980).

Motor vehicle emissions constitute the major source of lead emissions to the atmosphere, accounting for 88% of total lead emissions. The combustion of waste oil accounts for another 7%. As shown in Table 3.16, coal combustion is not a significant factor in total atmospheric lead emissions.

3.4.2 Summary of Health Effects

Studies of ambient air concentrations indicate that lead typically occurs in urban airborne suspended particles 0.5 μm or less in mass median equivalent diameter at annual average concentrations ranging from < 0.1-5 $\mu g/m^3$, with an overall average of 1-2 $\mu g/m^3$; however, urban concentrations of lead have declined somewhat since 1970. Lead concentrations in suspended particles

Table 3.14. Estimated Nationwide Carbon Monoxide Emissions, Selected Years from 1940 to 1975

Source Category	Emissions (10^6 metric tons/yr)[a]							
	1940	1950	1960	1968	1969	1970	1972	1975[b]
Transportation	31.7	50.2	75.7	102.5	101.6	100.6	70.4	66.5
Industrial process losses	13.1	17.1	16.1	7.7	10.9	10.3	15.8	13.3
Agricultural burning	8.3	9.4	11.2	12.6	12.5	12.5	1.5	0.8
Fuel combustion in stationary sources	5.6	5.1	2.4	1.8	1.6	0.7	1.1	1.3
Solid-waste disposal	1.6	2.4	4.6	7.3	7.2	6.6	4.5	3.4
Miscellaneous	17.2	9.1	5.8	4.9	5.7	4.1	4.2	1.7
Total	77.5	93.3	115.8	136.8	139.5	134.8	97.5	87.0

From National Academy of Sciences (1977c).

[a]To convert to U.S. short tons, multiply by 1.1 (1 short ton = 2×10^3 lb).

[b]Annual emission as of March 12, 1975.

Table 3.15. Levels of Carboxyhemoglobin and Reported Effects

COHb (%)	Effects	References[a]
0.4	Normal physiologic value for nonsmokers.	Cobwin et al. (1969); Sjostrand (1949)
2.5-3	Decreasee exercise performance in patients with angina or with intermittent claudication.	Anderson et al. (1973); Avonow et al. (1971, 1972, 1973)
4-5	Increased symptoms in traffic policeman (headache, lassitude).	Babayants (1962)
	Increased oxygen debt in nonsmokers.	Chevalier et al. (1966)
5-10	Changes in myocardial metabolism and possible impairment.	Ayres (1965, 1969)
	Statistically significant diminution of visual perception, manual dexterity, or ability to learn.	Bender et al. (1971); Halperin et al. (1959)
10+	Headache and impaired manual coordination.	Stewart et al. (1970)
	Changes in visual evoked response (VER) by EEG.	Hosko (1970)

From Ferris (1978).

[a]References cited are in the source and are omitted in this report.

Table 3.16. Estimated Atmospheric Lead Emissions for the United States, 1975

Source Category	Annual Emissions (10^6 ton/yr)	Emissions	
		As Percentage of Subtotal	As Percentage of Total
Mobile subtotal	142,000	100	-
Gasoline combustion	142,000	100	88.1
Stationary subtotal	19,225	100	-
Waste oil combustion	10,430	54.3	6.5
Solid waste incineration	1,630	8.5	1.0
Coal combustion	400	2.1	0.2
Oil combustion	100	0.5	0.1
Gray iron production	1,079	5.6	0.7
Iron and steel production	844	4.4	0.5
Secondary lead smelting	755	3.9	0.4
Primary copper smelting	619	3.2	0.4
Ore crushing and grinding	493	2.5	0.3
Primary lead smelting	400	2.1	0.2
Other metallurgical	272	1.4	0.2
Lead alkyl manufacture	1,014	5.3	0.6
Type metal	436	2.3	0.3
Portland cement production	313	1.6	0.2
Pigments	112	0.6	0.1
Miscellaneous	328	1.7	0.2
Total	161,225	-	100

From USEPA (1977).

in rural air samples range from < 0.01-1.4 $\mu g/m^3$, with an overall average of ~0.2 $\mu g/m^3$. Indoor concentrations of lead are variable, generally 1/3 to 2/3 the concentration of adjacent outdoor levels.

Routes of entry of lead into the body are inhalation of airborne lead and ingestion, both of food and non-food material. Inhaled concentrations are governed by the physical and chemical state of lead, particle size retention in the lung, and absorption from the lung into the red blood cells (see Section 4.1 on respirable particulates for additional information). Uptake is governed by the nutrient balance of food ingested, by the physical nature of the material ingested, and by absorption by circulating red blood cells.

Clinical studies on the deposition of airborne lead particulate matter in the human respiratory tract suggest that 30 ± 10% of the ambient air lead particulates that are inhaled will be deposited there. Of the lead thus deposited in the respiratory tract, it has been estimated that as much as 50% or more is absorbed and enters the blood stream. Lead also enters the bloodstream through ingestion resulting from exposure to leaded paint, lead in the diet introduced by processing, packaging, etc., and lead plumbing. The average daily dietary intake is about 300 µg.

Lead as a community air pollutant has been associated with increased blood lead in adults (Goldsmith and Hexter 1967; Hasselbald and Nelson 1975; Johnson et al. 1975) and in children (Landrigan et al. 1976; Goldsmith 1974). Most serious lead poisoning in children is due to ingestion of leaded paint from deteriorating housing. However, serious lead poisoning could result from prolonged breathing of air containing 3-5 $\mu g/m^3$ or more of lead (Berry et al. 1974). Lead is excreted by the kidneys or stored in bone, and acts on the constituents of blood, kidneys, and central nervous system. Early signs of poisoning include impairment of mental function, behavior problems, and anemia. At higher levels, lead in the system causes vomiting, cramps, and serious impairment of kidney and nervous system function. Anemia results from the action of lead on the red blood cells. Both the life span and average number of red blood cells are reduced. The mechanism of action of lead on the kidneys and central nervous system is not well understood.

Quantitative relationships pertaining to blood lead are available. Present evidence indicates that blood lead levels in humans follow a log-normal distribution. On the basis of log-normal distribution, it is possible to estimate the proportion of a population whose blood lead levels exceed any specified value. This information, along with the information in Table 3.17, which summarizes the hematological and neurological effects of lead, permits a risk assessment, i.e., evaluation of effects that relate to the general population. Laboratory animal studies on the neurobehavioral effects of lead provides further evidence of a differential sensitivity of newborn or young animals of many species to lead effects. These are extensively summarized and reviewed in USEPA (1977).

There have been substantially higher concentrations of atmospheric lead in the Los Angeles area than in other large cities, due to the large contribution of motor vehicle exhaust. In the Los Angeles area, higher blood lead concentrations are found for children and adults living in areas with heavier pollution.

Lead exerts effects on a broad range of subcellular systems. These effects have been reviewed by Goyer and Rhyne (1973) and Fowler (1978). In animals exposed to lead in vivo, evidence can be found of toxic effects at various subcellular sites including nuclei, mitochondria, endoplasmic reticulum and lysosomes (Fowler 1978). Deknudt et al. (1973) showed elevated frequencies of chromosome and chromatid abnormalities in circulating lymphocytes of men occupationally exposed to lead. Nuclei of kidney cells are known to develop inclusion bodies as well as higher rates of DNA synthesis after prolonged exposure to lead. Bone marrow cells from mice fed 1% lead acetate show significantly higher frequencies of chromosomal aberrations than cells from control animals. Mitochondrial swelling is an early indicator of kidney toxicity. Lead also inhibits mitochondrial enzymes involved with heme biosynthesis. Injection of lead results in decreased activity of the microsomal mixed-function oxygenases. These enzymes are involved in the detoxification of some polycyclic organic molecules, a prominent pollutant from coal combustion. Some studies have indicated that lead is concentrated in lysosomes (Fowler 1978).

In experiments with free-living, single-celled organisms, exposure to lead has resulted in several toxicological effects. Deknudt and Deminatti (1978) cultured lymphocytes from healthy human donors. In the presence of 0.01 M lead acetate, all cell division was blocked, while a ten-fold lower concentration allowed sufficient mitoses after 72 hours of cytogenetic analysis in their experiment. They failed to observe a significant increase in the frequency of chromosomal aberrations in the exposed cells. Shenker et al. (1977) studied the effects of lead chloride on B lymphocytes from B alb/c mice. After 72-hour exposure to concentrations ranging from 10^{-7} to 10^{-3} M, the fraction of viable cells, as determined by dye exclusion, was reduced at all doses with increasing toxicity with increasing concentration. If the cells were stimulted to divide, the effect was enhanced. Also the presence of lead caused increased uptake of both tritiated thymidine and tritiated alanine. This increased uptake was seen both in unstimulated and stimulated cells. Castranova et al. (1980) studied metabolism in rat alveolar macrophages. They determined the concentration of lead chloride that caused half maximal inhibition of both

Table 3.17. Blood Lead Levels versus Lowest-Observed-Effects Levels

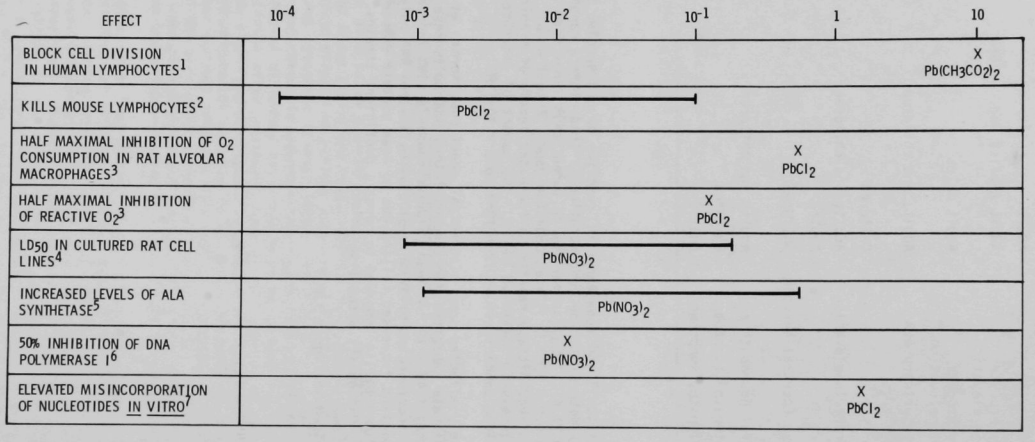
Lowest Level for Observed Effects (μg/Pb/dl whole blood)	Observed Effect	Population Group
10	ALAD inhibition	Adults and children
15-20	Free erythrocyte porphyrin elevation	Adult females and children
20-25	Free erythrocyte porphyrin elevation	Adult males
40	Increased urinary ALA excretion	Adults and children
40	Anemia	Children
40	Coproporphyrin elevation	Adults and children
50	Anemia	Adults
50-60	Cognitive (central nervous system)	Children
50-60	Peripheral neuropathies	Adults and children
80-100	Encephalopathy symptoms	Children
100-120	Encephalopathy symptoms	Adults

From USEPA (1977).

total oxygen consumption [5.6 ($\pm$1.3) $\times$ 10^{-4}] and appearance of reactive oxygen species [1.2 ($\pm$0.5) $\times$ 10^{-4}]. Kusell et al. (1978a) determined the concentration of lead nitrate capable of reducing reproductive survival by 50% (i.e., LD_{50}) in various cultured cells lines from rats; those values ranged from 8.5×10^{-7} to 1.8×10^{-4} M. In a companion paper, Kusell et al. (1978b) found that twenty-four-hour exposure to lead nitrate results in increased levels of δ-aminolevulinic acid synthetase activity compared to levels in unexposed cells. The amount of increase was relatively constant over the range of lead concentrations from 10^{-6} to 5×10^{-4} M.

In cell-free systems, lead has adversely affected some biochemical reactions. Korman et al. (1978) found that 1.1×10^{-5} M lead nitrate inhibited Micrococcus luteus DNA polymerase I by 50%. Sirover and Loeb (1976) found that 1.48×10^{-3} M lead chloride causes a >30% increase in the frequency of incorrect nucleotides incorporated by avian myeloblastosis virus DNA polymerase. In contrast, Nishioka (1975) failed to find increased killing due to lead acetate exposure in DNA-repair-deficient strains of Bacillus subtilis compared to normal, proficient strains. Such tests detect most chemicals that modify DNA or its metabolism.

The results discussed above are summarized in Figure 3.6.



1. DeKnudt and Deminatti (1978).
2. Shenker et al. (1977).
3. Castranova et al. (1980).
4. Kusell et al. (1978a).
5. Kusell et al. (1978b).
6. Korman et al. (1978).
7. Sirover and Loeb (1976).

Fig. 3.6. Concentrations of Lead (Pb) Causing Cellular Effects (mM).

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4. NONCRITERIA POLLUTANTS

4.1 RESPIRABLE PARTICULATES

4.1.1 Introduction

The inhalation hazards of particulate matter depend in large part on the concentration of deposits at sites or regions of the lung. Fine particles are chemically diverse, but their physical size is the main determinant of penetration and point of ultimate deposition in the lung (Lippmann 1977; Corn 1972; Amdur 1976; Yeh et al. 1976; Mercer 1973).

4.1.1.1 Regulatory and Physiological Definitions

The current air quality standard for total suspended particles (TSP), promulgated by the USEPA in 1971, was established without regard to particle size. Numerous research efforts initiated since passage of this standard have clarified the fundamental particle-size dependence of origin, transformation, chemical composition, and respiratory deposition. With this greater understanding of processes, Congress provided for a scientific review of national ambient air quality standards in the Clean Air Act Amendments of 1977. USEPA was charged with completing the review and promulgating revised standards by December 1980. The role of fine particulates in development of human respiratory disease has elicited much response from both the public and scientific community during USEPA's period of comment solicitation. A number of definitions and parameters have evolved from research and public discussion that will likely be incorporated into any revised particulate standard (Miller et al. 1979). These considerations will form a framework for generic analysis of potential health effects resulting from fine particulate exposures.

The common unit of size in most literature is the aerodynamic diameter, defined as the "diameter of a unit density sphere having the same settling velocity as the particle under consideration" (Morgan and Turner 1973). The use of this definition is a normalizing technique to accommodate the nonspherical nature of many airborne solids, liquids, and gases. They may be crystalline or amorphous forms, fibers, spheres, or aggregate meshes (Holland et al. 1979) on which transformation products, toxic elements, and organic molecules can adsorb.

Particles greater than 2 μ (aerodynamic diameter) arise from mechanical, abrasive forces and have been classified as "coarse" (Whitby and Cantrell 1976). These include materials such as sea spray and windblown dust. Most particles <2 μ are created during condensation of either hot or cold supersaturated vapors and are classified as "fine." These particles are produced largely during industrial processes. Note, however, that the variation of aerodynamic diameters found for normally "coarse" materials may extend into the "fine" range. Chemical and physical interactions between primary gases, liquids, and solids produced during condensation can result in transformation products identified on sample filters as coagulated or aggregated particles. Principal formation and removal mechanisms, as well as surface area distribution, of a domestic atmosphere aerosol are summarized in Figure 4.1.

Characteristics of physical deposition in biological systems also support a size discrimination between "fine" and "coarse" at approximately 2 μ (Fig. 4.2) (Lippmann 1977, Lippmann et al. 1979; Palmes and Lippmann 1977; Altshuler et al. 1957, 1967); linear measurements are used for diameters <0.5 μ since diffusional displacement is the dominant deposition mechanism for particles of this size--i.e. neither density nor shape is a significant parameter for very fine particles. At diameters <2.5 μ, total alveolar deposition ranges between 15 and 25%. Maximal deposition of approximately 45% occurs during mouth breathing by the same experimental group. Since alveoli are nonciliated and comprise the gas-exchange surface of the deep lung (Fig. 4.3), any material that is preferentially deposited in these structures requires careful scrutiny. This is particularly pertinent for particles <0.5 μ in linear diameter, which can undergo systemic transport in the body via diffusion. As a result of these findings, USEPA's Health Effects and Environmental Science Research Labs are recommending to the Office of Air Quality Planning and Standards a cut-off point of 2.5 μ for setting a size-specific standard.

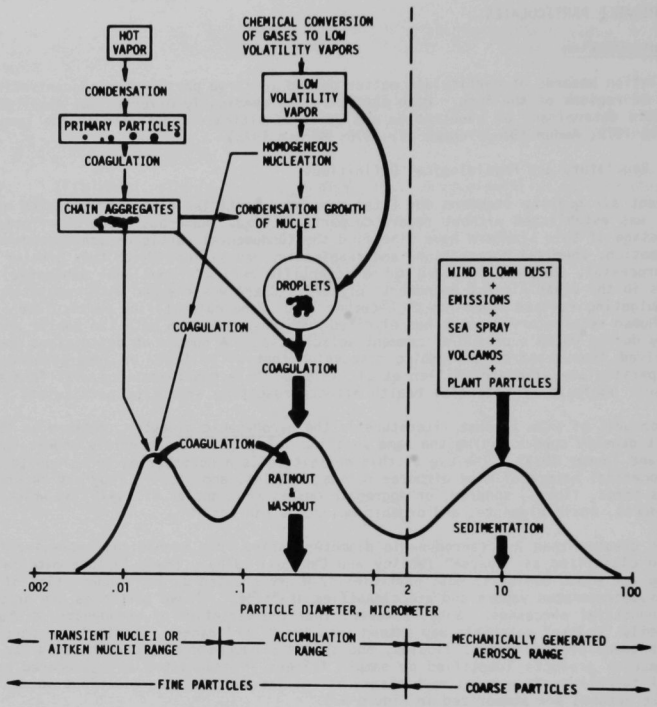


Fig. 4.1. Schematic of an Atmospheric Aerosol Surface Area Distribution Showing Principal Sources and Removal Mechanisms. Reproduced from Whitby and Cantrell (1976), Fig. 2, copyright 1976, IEEE.

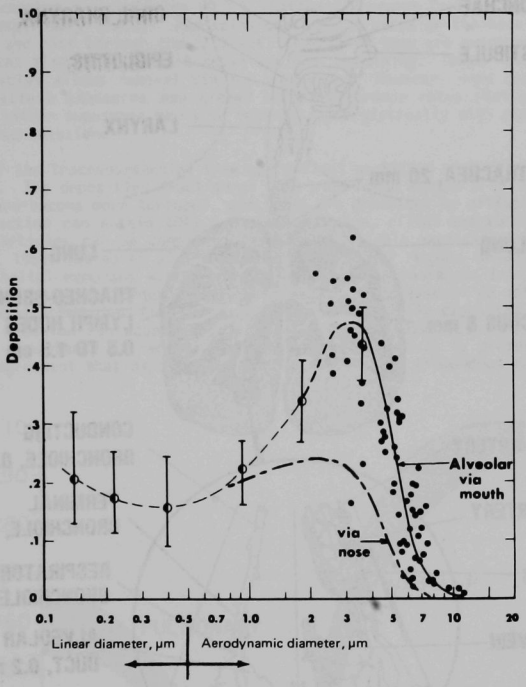


Fig. 4.2. Dependence of Alveolar Deposition on Particle Size and Route of Entry. Mouth-breathing plot derived by Lippmann et al. (1979, p. 124) from results by Lippmann (1977) and Palmes and Lippmann (1977). Nasal-breathing plot derived by Lippmann et al. (1979, p. 124). Reproduced from National Research Council (1979), p. 124, by permission of University Park Press.

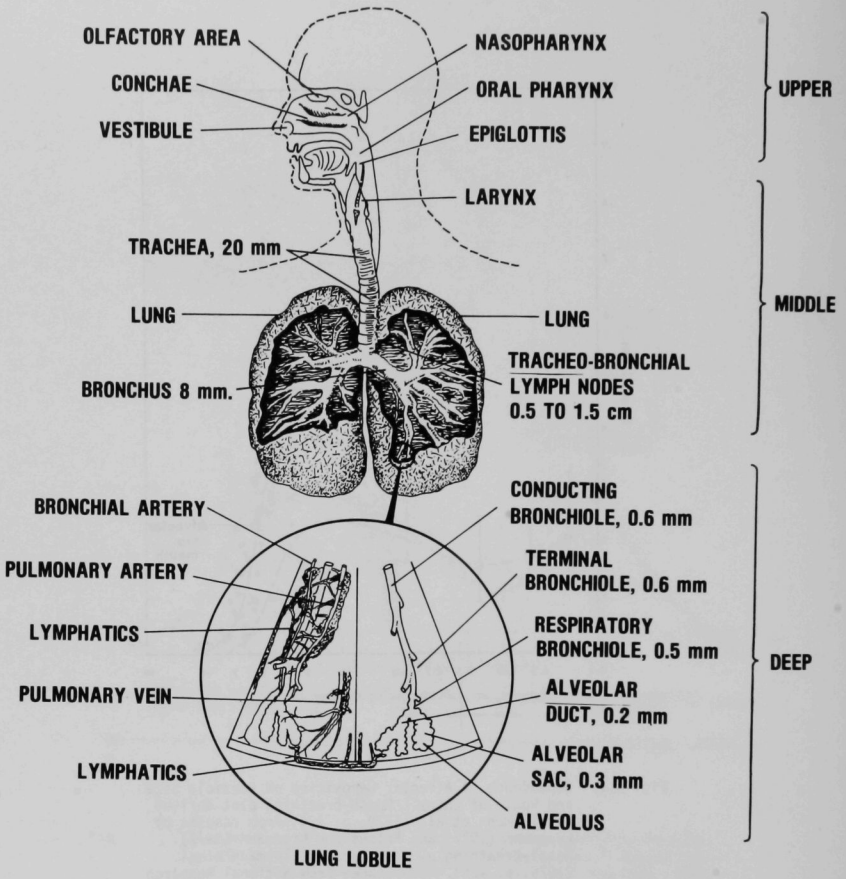


Fig. 4.3. Human Respiratory Airways. Redrawn from National Academy of Sciences (1961).

4.1.1.2 Deposition in Respiratory Passages

Most fine particles are hygroscopic and rapidly gain size and mass during passage through the humid atmosphere of respiratory airways. All larger particles, regardless of origin, may be removed from the airstream by several mechanisms. If deposited by impaction or sedimentation in the anterior nares, they are often mechanically removed by sneezing, nose blowing, etc. (National Research Council 1979). Once past the nares, particles <1.0 μ may impact or settle out on nasal hairs and bends of the conchae. Mucus transported by cilial action from the conchae to the anterior nares may carry material into the region of mechanical removal described above. Diffusion can also occur on the conchae for particles <0.1 μ (National Research Council 1979) (see Fig. 4.3 for illustration of respiratory tract). Particles captured in mucus on the naso- or oropharynx are quickly swallowed and pass through the GI tract, from which they are eventually eliminated. Laryngeal deposition can also occur in both nasal and oral breathing. Under normal conditions, mucus capture of particles allows removal via the esophagus. However, some occupational or domestic (i.e., cigarettes) exposures may exceed normal clearance rates (National Research Council 1979). These latter exposures may also interact synergistically with other nonoccupational or non-cigarette-related emissions.

The larger passages of the tracheobronchial tree may collect particles with diameters between 3 and 10 μ by impaction. The deposition fraction of this size range equals 2 to 12%. As airways decrease in diameter and become more tortuous, particles are deposited by diffusion and sedimentation (deposition fraction can attain 40%). In normal tissue, cilial and mucus clearance for inert and insoluble particles can be completed in one day; soluble particles are removed more quickly (Albert et al. 1969; Yeates et al. 1973). In the alveolar region, soluble particles may pass through the epithelial membrane within minutes. Half-time clearances for insoluble particles in this zone may be on the order of months or years, since alveoli do not contain cilia or mucus. It is to be expected that fine particles are preferentially deposited in this region, where the deposition fraction can equal 65% (Task Group on Lung Dynamics 1966) (Fig. 4.4). Although the relative importance of removal mechanisms in the alveoli is presently unclear, there is substantial agreement that deposition in these tissues has tremendous damage potential.

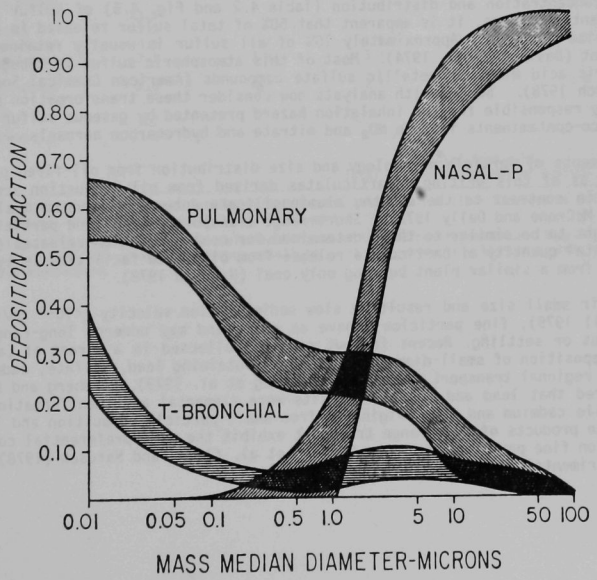


Fig. 4.4. Fractional Deposition versus Aerodynamic Diameter. Tidal volume of 1450 mL^3. Each of the shaded areas is an envelope indicating the variability of deposition for a given mass median aerodynamic diameter in each compartment. From Task Group on Lung Dynamics (1966).

4.1.1.3 Composition and Co-Contaminants

Particulate matter can be classified according to origin. Primary particles, those injected directly into the atmosphere, come mainly from combustion and industrial processes, incineration and refuse burning, and transportation systems. Secondary particles, those formed from atmospheric chemical reactions, are made up largely of sulfates and organic matter. The sources of these types of emissions and the importance of coal combustion are indicated in Table 4.1; coal-burning electric utilities alone produce almost 75% of the fine particles from fuel combustion (Shannon et al. 1974).

A number of toxic elements released to the atmosphere during coal combustion for power generation are found preferentially associated with small particle emissions. It is thought that these elements and/or their compounds volatilize during combustion and condense or adsorb onto the fine particles that are not captured by existing precipators or flue-gas desulfurizers (Natusch et al. 1974). Those elements in airborne fly ash known to exhibit pronounced trends of increasing concentration with decreasing diameter are listed in Table 4.2 (Davison et al. 1974). Similar inverse relationships between fly ash particle size and element concentration have been noted by Lee and von Lehmden (1973) and Toca (1972). Other elements found in fine fly ash include iron, manganese, vandium, silocon, magnesium, calcium, beryllium, and aluminum; these exhibit only limited concentration trends (Davison et al. 1974).

Some compounds found concentrated on the surfaces of fine particles are not only known to be toxic, but are also suspected carcinogens. Particulate polycyclic organic matter (POM) and compounds of the elements lead, cadmium, selenium, arsenic, nickel, and chromium fall into this latter category (Natusch 1978). The particle-size dependencies of arsenic, nickel, cadmium, and sulfur concentrations are clearly illustrated in Figure 4.5. High concentrations of all elements listed above, particularly sulfur, are found in a 300-angstrom-thick shell on fly ash surfaces (Natusch 1978). This ready availability of toxic elements and large surface area provided by fine particles allows intimate contact with body fluids and alveolar tissues (Natusch 1978). As a result, there is legitimate concern that the manifest health effects of exposure may be systemic rather than respiratory alone (U.S. Office of Technology Assessment 1979).

By comparing concentration and distribution (Table 4.2 and Fig. 4.5) of sulfur in monitored (coal) powerplant emissions, it is apparent that 50% of total sulfur released is associated with the smallest size fraction. Approximately 10% of all sulfur is usually retained as fly ash within the plant (Davison et al. 1974). Most of this atmospheric sulfur is thought to be in the form of sulfuric acid mists or metallic sulfate compounds (American Chemical Society 1969 as cited in Natusch 1978). Some health analysts now consider these transformation products to be at least partly responsible for the inhalation hazard presented by gaseous sulfur oxides (Natusch 1978). Other co-contaminants include NO_2 and nitrate and hydrocarbon aerosols.

Similar assessments of particle morphology and size distribution from oil-fired plants have not been published as of this writing. Particulates derived from oil combustion are known to be highly porous in contrast to the smooth, aluminosilicate spheres common to coal combustion (Natusch 1978; McCrone and Delly 1973). Short-range transport (<8 km) and particle-size distributions are thought to be similar to those determined for coal combustion releases (Natusch 1978). However, the total quantity of particulate release from oil-fired facilities is typically 50 times less than that from a similar plant burning only coal (Natusch 1978).

Because of their small size and resulting slow sedimentation velocity ($<10^{-2}$ cm/sec; National Research Council 1979), fine particles behave as gases and may undergo long-range transport prior to washout or settling. Recent fallout samples collected in a forest of East Tennessee suggest that deposition of small-diameter particles containing lead, sulfate, cadmium, and zinc is a result of regional transport phenomena (Lindberg et al. 1979). Lindberg and his colleagues (1979) discovered that lead and sulfate deposits were elemental and transformation products of combustion, while cadmium and zinc originated from small-particle combustion and large-particle disperson. These products of long-range transport exhibit the same preferential concentration of toxic elements on fine particles noted by Davison et al. (1974) and Natusch (1978). A sample of Lindberg's experimental results is presented in Table 4.3.

Table 4.1. Fine Particle Emissions from Industrial Sources (mass basis, 10^3 ton/yr)

Source	Fine Particle Size Range (μm) 1-3	0.5-1.0	0.1-0.5	0.05-0.1	0.01-0.05	Total
Stationary combustion						
Coal						
Electric utility	661.5	198.2	108.7	3.1		971.5
Industrial	85.2	14.5	6.2	0.1		106.0
Fuel oil						
Electric utility and industrial	126.9					126.9
Natural gas and LPG						
Electric utility and industrial	97.2					97.2
				Subtotal--fuel oil and gas		224.1
				Total from all fuel combustion		1,301.6
Crushed stone	868.0					868.0
Iron and steel	68.5-88.5	42.6-78.6	168.0-393.5	2.4-24.3	1.5-5.1	283-590.0
Kraft pulp mills	146.0	90.1	81.2	1.7		319.0
Cement plants, rotary kilns	130.8	32.7	13.5			177.0
Hot-mix asphalt plants	110.8	38.0	21.7			170.5
Ferroalloys	19.2	27.8	81.1	17.3	7.7	153.1
Lime plants	65.6-139.6	18.8	23.6	3.0	1.8	(113.0 186.8)
Secondary nonferrous metallurgy	127.0					127.0
Carbon black	93.0					93.0
Coal preparation plants, thermal dryer	63.5					63.5
Petroleum FCC units	45.0					45.0
Municipal incinerators	10.4	6.7	11.5	3.5	4.3	36.4
Fertilizer, granulators and dryers	7.1	3.5	3.1			13.7
Iron foundries, cupolas	6.8	2.4	3.1	0.4	0.4	13.1
Acids						
Sulfuric	2.7					2.7
Phosphoric (thermal)	1.0					1.0
				Total from acids		3.7
				Total from major industrial sources		3800-4142

From Shannon et al. (1974) (based on "Particulate Pollutant Systems Study, Volume III - Handbook of Emission Properties," Midwest Research Institute, EPA contract No. CPA 22-69-104, August 1, 1971).

Note: Potentially significant sources were not evaluated because of lack of sufficient data. They are: (1) operations related to agriculture, (2) primary nonferrous metallurgy, (3) clay products, (4) food processing operations, and (5) fiberglass manufacture.

Table 4.2. Concentration of Toxic Elements in Airborne Fly Ash[a]

Particle Diameter (μm)	Elemental Concentration (μg/g)									
	Pb[b]	Tl[c]	Sb[c]	Cd[b]	Se[b]	As[b]	Ni[b]	Cr[b]	Zn[c]	S[d] (wt. %)
>11.3	1,100	29	17	13	13	680	460	740	8,100	8.3
7.3-11.3	1,200	40	27	15	11	800	400	290	9,000	-
4.7- 7.3	1,500	62	34	18	16	1,000	440	460	6,600	7.9
3.3- 4.7	1,550	67	34	22	16	900	540	470	3,800	-
2.1- 3.3	1,500	65	37	26	19	1,200	900	1,500	15,000	25.0
1.1- 2.1	1,600	76	53	35	59	1,700	1,600	3,300	13,000	-
0.65- 1.1	-	-	-	-	-	-	-	-	-	48.8

[a]Adapted from Davison et al., 1974 (Table 1, p. 1109).

[b]Spark source mass spectrometry.

[c]DC arc emission spectrometry.

[d]X-ray fluorescence spectrometry.

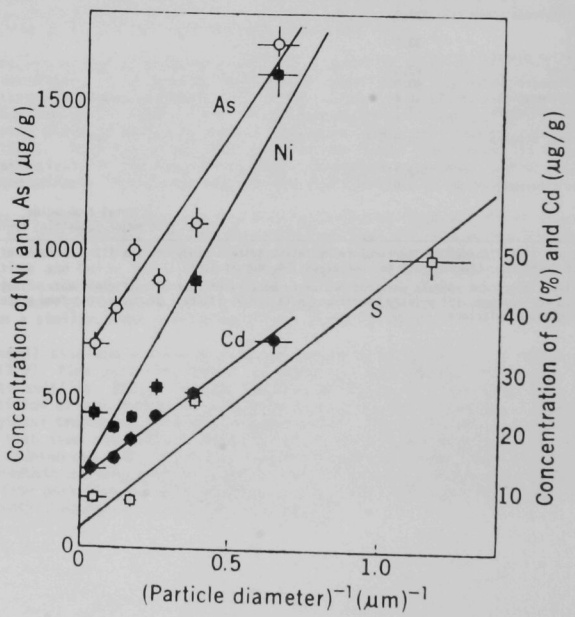


Fig. 4.5. Dependence of the Average Concentrations of Arsenic, Nickel, Cadmium, and Sulfur on Airborne Particle Size in Coal Fly Ash. From Natusch et al. (1974). Copyright 1974 by the American Association for the Advancement of Science.

Table 4.3. Some "Available" Metal[a] and Sulfate Air Concentrations in Walker Branch Watershed (Roane County, Tennessee) during Intensive Sampling Experiments[b]

Experimental Period	Aerodynamic[c] Diameter (μ)	Total Suspended Particulates (TSP)	Cadmium (Cd)	Lead (Pb)	Zinc (Zn)	Sulfate ($SO_4^=$)
W1	Total	45 μg/m³	0.12 ng/m³	92 ng/m³	3.5 ng/m³	11.8 μg/m³
	> 8.0	17%	18%	2%	14%	2%
	3.7	25	33	7	16	2
	1.2	12	18	8	19	3
	0.5	13	18	16	18	22
	< 0.5	32	13	67	35	71
W2	Total	64 μg/m³	0.24 ng/m³	170 ng/m³	7.4 ng/m³	18.0 μg/m³
	> 7.8	18%	6%	1%	8%	1%
	3.7	17	16	4	7	2
	1.2	12	23	8	11	8
	0.48	30	10	33	8	48
	< 0.48	24	44	53	66	42

[a]"Available" metal = distilled leachable plus dilute acid leachable.

[b]Adapted from Lindberg et al. (1979).

[c]Assuming a 50% particle collection efficiency.

4.1.2 Summary of Health Effects

Studies of fine particulate exposure in healthy and asthmatic individuals have been performed with inert dusts and powders such as $CaCO_3$. Functional changes (i.e., airway flow resistance, gas trapping, and abnormal intrapulmonary mixing) have been observed after brief inhalation of particles <1 μ in diameter (Dubois and Dautrebande 1958; Lovejoy et al. 1961). These changes may be caused by edema, excessive mucus secretion, or constriction of smooth muscle, trachea, or bronchii (Amdur and Underhill 1968). The exact mechanism is unknown. The additional alteration of impaired gas exchange also was noted (Lovejoy et al. 1961). An obvious difference between the two populations was length of the lag time between onset of exposure and observed responses. The lag was, of course, greater in healthy subjects.

Epidemiologic evaluations have been confined largely to major, acute episodes where large numbers of people were stricken. The Donora, New York City, Meuse Valley, and London episodes are discussed in Section 3. At the time of these incidents, both investigators and instrumentation were insensitive to the potential role played by respirable particles in mortality and morbidity induction. Perhaps the data collected during the recent 10-year monitoring program initiated by the Harvard School of Public Health in six communities throughout the United States will clarify the potential linkages between fine particles and human health (Ember 1977). The best available evidence indicates that inhalation exposure to fine particles is likely harmful and may have adverse health effects. Possible biological responses following deposition of particles in the respiratory tract are summarized in Table 4.4 (Shannon et al. 1974; Corn 1972; Perera and Ahmed 1978).

Table 4.4. Possible Effects Produced by Inhaled Particulate Matter after Deposition in Respiratory Tract Compartments

Compartment in which Deposition Occurs	Soluble Particle	Insoluble Particle
Upper	Damage to mucosa and paralysis of cilia.	Transferred to gastrointestinal tract.
Nasopharyngeal	Allergic response.	
		Removed with sputum.
		Allergic response.
Middle	Reflex bronchoconstriction.	Short-term clearance to gastrointestinal tract.
Tracheobronchial	Allergic response.	
		Removed with sputum.
	Damage to mucosa and paralysis of cilia.	
	Susceptibility to infection.	
	Potentiation if gas (SO_2, NO_2, O_3, etc.) exposure present.	
Deep	Damage to alveolar epithelium.	Long-term retention: react with tissue to cause local effects.
Pulmonary	Peripheral respiratory unit constriction.	Remain in tissue (inert).
	Potentiation if gas (SO_2, NO_2, O_3, etc.) exposure present. May lead to release of proteolytic enzymes and eventual emphysema with alveolar destruction.	Transported to lymph nodes.
		Short-term retention: Phagocytized and transported to terminal bronchioles with subsequent clearance from tracheobronchial compartment.

From Shannon et al. (1974), Corn (1972), and Perera and Ahmed (1978).

Since 1924, the use of electrostatic precipitators (ESPs) has been the method of choice in controlling particulate releases from coal-fired boilers (Manhattan College Dept. of Chemical Engineering (MCDCE) 1978). This method has been highly successful at reducing large-diameter emissions, but is inherently less efficient at capturing fine ash. Even though fine particles do not represent the greatest mass released from coal-fired facilities, the portion <2.5 μ is, nevertheless, considerable. Approximately 30% of the total release falls in this size class even with installation of electrostatic precipitator controls (Lee et al. 1975). The distribution of aerodynamic diameter and cumulative mass for emissions from one ESP-controlled Midwestern generating plant is illustrated in Figure 4.6. Such a distribution is considered common.

Wet scrubbers can effectively remove both gaseous and particulate pollutants from stack gases by inertial impaction, direct interception, diffusion, and condensation (MCDCE 1978). Collection of SO_2 and nitrogen oxides by this method removes sulfate and nitrate precursors from the stack release, and can, therefore, reduce the rate of secondary combustion product formation. Various wet scrubber designs are used, but only the variable-throat Venturi has been commercially effective at removing fine particles from boiler plant combustion gases. For very small particle diameters, collection efficiency of wet scrubbers is much less than 40% (MCDCE 1978).

The most efficient method for fine-particle control is the fabric filter, which can provide high collection efficiencies for particles of 0.5 μ diameter and reasonable control of ultrafine materials ≤0.1 μ (MCDCE 1978). Baghouses of woven glass fiber filters are most often used in conjunction with coal-fired boilers, with flue gas being drawn through the bag from inside. Fine particle collection efficiencies of several glass fiber filter baghouse designs have exceeded 99% for all common boilers (MCDCE 1978).

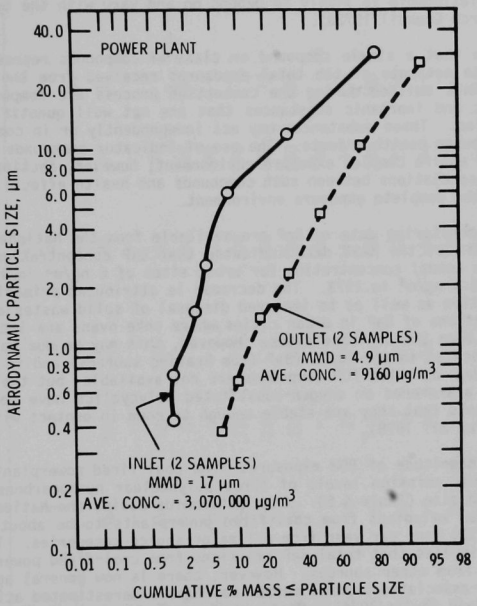


Fig. 4.6. Composite Mass Size Distribution of Particulates Collected before and after an Electrostatic Precipitator at a Coal-Fired Powerplant. From Lee et al. (1975). Reprinted with permission from Environmental Science and Technology 9(7):643-647. Copyright 1975, American Chemical Society.

4.2 ORGANIC MATTER

4.2.1 Introduction

Polycyclic organic matter (POM) may be formed during the combustion or pyrolysis of fossil fuels or other materials containing carbon and hydrogen. While POM can include many chemical groups, the two classes of POM compounds of major concern that are found most often in ambient air are polynuclear aromatic hydrocarbons (PNAs) and their neutral nitrogen analogues (Santodonato et al. 1979). These two chemical groups have been shown to contain a number of carcinogenic agents (National Research Council 1972a). As a consequence, the PNAs, and more specifically the potent carcinogen benzo(a)pyrene (BaP), have been the most extensively studied POM compounds.

At present, BaP concentrations in the atmosphere provide the most widely available indicator of PNAs. BaP's usefulness as as indicator of other PNAs and of total carcinogenicity is not fully established. While some investigators have found good correlations between BaP and other PNAs (Sawicki 1967), this relationship is likely to depend on and vary with the types of POM emission sources (National Research Council 1972a).

It is important to note that a single compound or class of compounds represents a relatively imprecise and incomplete estimate of the total exposures received from the process of coal combustion. The effluents emitted during the combustion process are composed of a complex mixture of both organic and inorganic substances that are not well quantified, and in many cases, not well qualified. These substances may act independently or in combination to influence the occurrence of human health effects. The use of indicator compounds provides a starting point for assessing the entire complex exposure environment; however, caution must be used when interpreting possible associations between such compounds and health effects since they do not, in most cases, reflect the complete exposure environment.

The most comprehensive monitoring data on BaP are available from the National Air Surveillance Network (NASN). Analysis of the NASN data indicates that BaP concentrations have declined steadily with an average annual concentration for urban sites of 6 ng/m^3 in 1958 (Sawicki 1967), 3.2 ng/m^3 in 1966, and 0.5 ng/m^3 in 1973. The decrease is attributed primarily to decreases in residential coal combustion as well as to improved disposal of solid wastes and restrictions on open burning. Concentrations of BaP in urban cities where coke ovens are located were considerably higher (40 to 70%) than the non-coke sites. However, this may be due to the fact that most of the coke ovens are located in the north (BaP from heating sources) and in highly industrialized areas. Recent trends on other POM compounds are not available, but the concentrations of at least 25 PAH and 32 aza-arenes or oxygen-substituted polycyclics have been determined in ambient air, which suggests that they are stable enough to come in contact with humans, animals, and plants (Santodonato et al. 1979).

Some data exist on the magnitude of POM exposures from coal-fired powerplants. Diehl et al. (1967) have determined the emission levels of three polynuclear hydrocarbons from coal-fired units of varying type and size (Table 4.5). On a nationwide basis, the National Research Council (1972a) has estimated BaP emissions from coal-fired powerplants to be about 1 ton per year compared to a total of 1320 tons per year from all major source categories. The National Research Council ratio of 1:1320 implies that total BaP emissions from coal-fired powerplants are neglible compared with emissions from other sources. However, there is now general agreement that past measurements of particle-associated organic matter may have underestimated actual concentrations (Natusch 1978; Van Hook and Shults 1976). Most organic emissions are in the vapor phase within stacks and do not condense onto the surfaces of fly ash particles until the stack plume has cooled to 100-200°F, some distance from the stack mouth. Thus, older measurements using in-stack collection methods of fly ash sampling may reflect only a small fraction of the total organic material emitted.

The association between POM and particles in the atmosphere is important when considering health effects. Many studies indicate that BaP is primarily associated with particulate matter, the great majority of which is in the respirable size range, about 0.1 to 2 μm in diameter (Santodonato et al. 1979). (POM preferentially condenses onto small particles because of their larger surface-to-volume ratio.) Thus, almost all the POM emitted from coal-fired powerplants is capable of pulmonary deposition, given the joint presence of small particles upon which it may be absorbed (see Sec. 4.1, Respirable Particulates).

Assessing possible human health effects from exposure to POM emissions, especially at low levels, is problematic for a number of reasons. Many of the data are from the occupational setting where exposures are considerably higher than those expected from coal combustion. Also, both community and occupational data reflect exposures to a variety of different POM-containing mixtures of compounds that may be quite different from the mixture of exposures from coal combustion.

Finally, the great majority of studies incorporate no actual measures of exposures, so that the exposure variable is usually some qualitative category (e.g., urban versus rural) or an estimate made retrospectively.

Table 4.5. Polynuclear Hydrocarbon Concentrations in the Flue Gases of Coal-Fired Power Units

Type of Burner	Capacity (lb steam/hr)	Load factor sampled	Benzo(a)pyrene			Benzo(e)pyrene			Benz(a)anthracene		
			μg/1000 m^3	μg/10^6 Btu	mg/hr	μg/100 m^3	μg/10^6 Btu	mg/hr	μg/1000 m^3	μg/10^6 Btu	mg/hr
Chain grate	33,000	0.61	-	-	-	250	119	3.5	-	-	-
Chain grate	35,000	0.45	9	4	0.1	91	42	1.0	44	20	0.4
Chain grate	40,000	0.47	110	75	2.8	440	300	11	-	-	-
Chain grate	52,000	0.41	850	500	20	530	310	12	1,400	820	32
Chain grate	55,000	0.81[a]	-	-	-	-	-	-	-	-	-
Underfeed	160,000	0.74	240	120	13	140	72	7.7	380	200	21
Underfeed	25,000	0.40[a]	53	31	1.1	590	350	12	-	-	-
Spreader	24,000	0.75	120	68	1.3	820	470	9.1	-	-	-
Pulsating grate	2,470	1.00	650	330	1.3	1,300	670	2.7	1,300	670	2.7
Pulverized coal	40,000	0.80	420	230	9.1	860	470	19	400	220	8.7
Pulverized coal	240,000	0.88	180	120	45	1,300	880	330	-	-	-
Pulverized coal	600,000	1.10	-	-	-	160	64	0.06	-	-	-
Pulverized coal	830,000	1.05	66	21	31	110	34	50	-	-	-
Pulverized coal	1,060,000	1.00	18	6	10	380	130	220	-	-	-
Pulverized coal	1,250,000	1.02	80	32	59	68	27	50	-	-	-
Pulverized coal	2,030,000	0.78	59	20	54	130	44	120	-	-	-
Pulverized coal	2,100,000	0.85	56	17	28	120	37	62	-	-	-
Cyclone	2,200,000	0.99	40	16	49	140	55	170	-	-	-

From Diehl et al. (1967).

[a]Fluctuating load.

The following sections are a review of the available literature concerning human health effects associated with POM at a variety of exposure levels. An attempt has been made to focus on those situations where some measurement or reasonable estimate of exposure is possible.

4.2.2 Summary of Health Effects

4.2.2.1 Acute Health Effects

Exposure to relatively high levels of POM-containing substances has resulted in various non-neoplastic skin and eye responses in a number of clinical and occupational settings. Skin effects have been produced in the clinical setting through the application of coal tar and coal tar solutions to the skin of human subjects, often in conjunction with exposure to ultraviolet light. Responses to this type of exposure include tar-phototoxicity, or erythema (Tanenbaum et al. 1975; Kaidbey and Kligman 1977), decreased mitotic activity (Fisher and Maibach 1973), and induction of cutaneous aryl hydrocarbon hydroxylase (AHH), a carcinogen-metabolizing enzyme that may induce cancer (Bickers and Kappas 1978).

Non-neoplastic skin effects also have been observed in occupational settings. These include nonallergic and allergic dermatitis, phototoxicity and photoallergic reactions, folliculitis, acne, and pigment disturbances. Effects have occurred following exposure to coal tar and coal tar products, pitch, creosote, asphalt and petroleum products (National Research Council 1972).

Additional sources have reported the occurrence of acute eye effects following exposure to coal tar pitch, including inflammation, conjunctivitis, and reduction in visual acuity (Emmett et al. 1977).

4.2.2.2 Occupational Neoplastic Disease

Skin carcinomas have historically been observed in occupations having exposure to high-temperature coal tar products. Much of the literature describes case histories and small groups of cases (Santodonato et al. 1979; National Research Council 1972b although some larger studies do exist (Henry 1947). In these studies, no quantification of exposure is available and comparison or control groups typically were not incorporated.

A number of epidemiological studies of occupational groups exposed to POM have been reported, many among coke plant workers and gas production workers and others from the roofing and asphalt industries (Table 4.6). The majority of these studies have shown that long-term exposure to the products of coal distillation is associated with an elevated rate of lung cancer, and in some cases, cancer of other sites.

Evidence supporting an increased risk of cancer mortality among workers in gas generation facilities comes from studies conducted in Japan (Kawai et al. 1967) and Great Britain (Doll et al. 1965, 1972). Japanese workers exposed to emissions of tarry fumes exhibited an occurrence of lung cancer mortality 33 times above that expected (a 3200% excess) based on a control group of steel workers who had never been employed at the gas generator plant. The risk of lung cancer increased with increasing duration of employment. The investigators concluded that smoking history and socioeconomic status were similar in the exposed and unexposed groups and could not account for the excess lung cancer risk observed. Unfortunately, no information on exposure levels is available for this cohort.

A group of British gas workers who had been employed by the industry at least five years were divided into three exposure categories and followed for a period of eight years to determine differences in mortality (Doll et al. 1965). Workers were categorized as having heavy exposure in carbonization plants, intermittent exposure, or no exposure. The mortality rate for lung cancer in those workers with the greatest exposure was 69% greater for lung cancer and 300% greater for bladder cancer than the mortality rates in the employed group with no exposure. An additional four years of followup (Doll et al. 1972) confirmed the excess lung and bladder cancer risk and also revealed an elevated risk of scrotal cancer among the exposed. Work as a topman appeared to be particularly hazardous with respect to all these cancers because of exposure to higher concentrations.

An estimation of the exposure incurred by the British gas workers can be made using data collected by Lawther et al. (1965). Lawther's measurements indicate an average BaP concentration of 3,000 ng/m^3 in English gas works with concentrations slightly over 200,000 ng/m^3 above the old horizontal retort houses. While these measurements may not accurately reflect the exact exposure conditions experienced by Doll's cohort (or by the Japanese gas workers), they represent the best available BaP exposure data for workers in gas generating facilities.

Table 4.6. Summary of Epidemiological Evidence of Carcinogenicity in Occupational Groups

Investigator	Date of Study	Type of Study	Site	Results	Characterization of Exposure
Kuroda and Kawahata	1936	Historical perspective	Lung	(12 lung cancer deaths out of a total of 15 deaths for all cancers). Relative risks cannot be calculated but there is suggestion of 26-fold excess over general population (Lloyd 1971).	Producer gas workers.
Kawai, Amamoto and Harada	1967	Historical perspective	Lung	33 times the rate for other steelworkers.	Workers in producer gas works.
Henry, Kennaway, and Kennaway	1931	Historical perspective	Bladder	1-1/2 to 4-fold increased risk relative to general population.	Workers in 5 out of 10 occupations involving exposures to coal products.
Kennaway and Kennaway	1936	Historical perspective	Lung	3-fold excess for "gas stokers and coke-oven chargers".	British producer gas workers; chimney sweeps and several categories of gas works employees.
Kennaway and Kennaway	1947	Historical perspective	Lung		
Doll	1952	Historical perspective	Lung	81% excess of lung cancer deaths.	Former gas retort workers (pensioners).
Doll	1965	8-year perspective	Lung	Those with heavy exposure showed 69% excess relative to those with minimal exposure (by-products workers).	Gas-worker mortality was greatest for those with greatest exposure (retort house workers).
			Bladder	Those with heavy exposure showed 4-fold excess reltive to those with minimal exposure.	
Doll	1972	4-year follow-up of 8-year perspective	Lung	Heavily exposed workers showed highly significant excess; by-products workers showed no excess.	
Reid and Buck	1956	Retrospective study of deaths among coke plant workers during 1949-1954	Lung	No excess in number of cancer deaths among coke plant workers as a whole nor of lung cancer for oven workers.	Coke plant workers.
Brunsgaard	1959	Retrospective study of deaths among employed and retired gas-workers over a 15-year period	Lung	6.4-fold increase over general population.	All deaths occurred among gas-workers with at least 5 years experience; most had more than 10 years.
			Bladder	5 deaths that appeared to be a significant excess.	

Table 4.6. (concluded)

Investigator	Date of Study	Type of Study	Site	Results	Characterization of Exposure
Lloyd, Lundin, Redmond, and	1970	Perspective study of mortality among steel-workers over work-area	Lung	Coke plant workers had 2-fold excess relative to rate among steelworkers.	Coke plant workers.
Lloyd	1971	Perspective study	Lung	Coke-oven workers showed 2-1/2-fold excess over steelworker population.	Coke plant workers.
			Pancreas	Top-side workers had 5-fold risk; top-side workers with 5 years full-time.	
			Intestine	Top-side had 10-fold risk; nonoven workers may have excess risk of digestive cancer.	
Redmond, Ciocco, Lloyd, and Rush	1972	Follow-up to perspective study	Lung	Coke oven workers had 1.34 excess risk relative to nonoven workers.	Coke plant workers
			Kidney	Coke oven workers had a 7.49 excess risk over nonoven workers.	
			Prostate	Coke oven workers had 1.64 excess risk over nonoven workers.	
Redmond	1976	Follow-up to previous study	Lung	15.72 excess for full-time top-side oven workers	
			Intestine	2.37 excess for nonoven workers.	
			Pancreas	4.29 excess for nonoven workers.	
Redmond et al.	1976	Historical perspective of by-products workers	Kidney	5 excess for all coke plant workers.	
			Lung	3.31 excess for oven workers.	
			Pancreas	4.95 excess for nonoven workers.	
			Intestine	2.93 excess for nonoven workers.	
			Buccal Pharyngeal		

From Santodonato et al. (1979). References cited are in the source and are omitted in this report.

Coke oven workers constitute another occupational group that has historically been exposed to high levels of POM. In a series of studies conducted in the United States, Lloyd (1971) and Redmond et al. (1972, 1976) compared the mortality experience of men employed in the coking industry to that of all steelworkers. Those employees working exclusively at the top of the ovens for five or more years had a 900% excess lung cancer mortality. Men working at both the side and topside of the coke ovens had a 186% excess lung cancer mortality, while those exposed only at the side of the ovens had a 46% excess lung cancer mortality relative to all steelworkers. In addition, an elevated risk of kidney cancer mortality (469% excess) was observed among all oven workers employed five years or more.

A number of studies have been conducted to determine BaP concentration existing in coke oven facilities. Bridbord et al. (1976) indicate that the average exposure for workers at the topside of the coke ovens is 18,000 ng/m^3 and about 7,000 ng/m^3 at the side and bench area. Jackson et al. (1974) and Masek (1971) give a range of measurements for top of battery locations, 1,200-15,900 ng/m^3 and 1,300-42,700 ng/m^3, respectively. Again, it is difficult to gauge the direct applicability of these exposure concentrations to the actual exposures experienced by the Lloyd and Redmond et al. cohorts. The exposures of interest in this occupational group are those exposure conditions in existence years prior to the occurrence of the excess cancers. Whether the relatively current measurements referenced above reflect those conditions is not clear.

A study conducted by Hammond et al. (1976) showed workers in the roofing and waterproofing industry to be at excess risk for cancers of many sites. When compared to the general U.S. male population, employees with at least 20 years of work experience had 59% excess risk of lung cancer, a 68% excess risk for both bladder cancer and leukemia, and a 300% excess risk of skin cancer (excluding melanoma). Those workers employed 40 years or more had a 147% excess risk of lung cancer.

To obtain exposure information for roofers and waterproofers, a sample of men working in various jobs wore masks during an entire work shift and the material collected in the masks was then analyzed. The average BaP concentration on the mask filters was 16,700 ng per 7-hour working day with a range of 1,400 to 53,000 ng BaP for the different jobs. The authors noted that a worker usually took turns at the various tasks rather than specializing in any one job. Assuming an average breathing rate of 8 m^3 per 7-hour working day and assuming that all BaP in the ambient air is deposited on the mask filter, the 16,700 ng BaP mask measurement can be converted to an equivalent ambient air concentration of 2088 ng/m^3 BaP. This value is considerably lower than air measurements made by Sawicki (1967) in the vicinity of pitch roofing operations (14,000 ng/m^3) and coal tar pitch working areas (75,000 ng/m^3).

The development of consistent dose response relationships from the occupational data presented above is difficult, given the wide range in exposure estimates and in observed mortality. To facilitate interpretation, these data are arranged along a BaP dose continuum in Figure 4.7. Discrepancies in the health effects occurring at different BaP levels probably reflect the limitations in the available exposure data and the inherent inadequacy of using one substance--BaP in this case--as a surrogate for the complex and varying ambient conditions to which these groups were exposed.

4.2.2.3 Community Studies

Community air pollution studies as well as occupational studies provide evidence on the health effects of POM. These studies typically involve much lower levels of ambient exposure, and are investigations of possible associations between community mortality and morbidity rates and some direct or indirect measure of air pollution.

Community air pollution studies are problematic for a number of reasons. In most cases, there is no information on or control of smoking and occupational exposures. Smoking is the major etiology agent in lung cancer, and the influence of both smoking and occupation in the occurrence of cancer outweigh any effect caused by air pollution. In addition, community studies often do not include estimates of the magnitude and duration of exposure. When pollutant measurements are available, these measurements probably do not reflect the exposures of interest, i.e., the exposures that occurred at an earlier time and are responsible for initiation of the cancer. Finally there is the problem of identifying the specific agent or agents that may be responsible for an increase in disease. As in occupational studies, BaP is most often used as an indicator of other POM. However, it should be considered an imperfect indicator since the proportion of BaP to other POM varies with the types of POM emissions sources contributing to the ambient atmosphere (National Research Council 1972b). Also, the carcinogenic effects of pollutant mixtures such as air pollution and cigarette smoke are not fully explained when BaP is used (Kotin et al. 1954; Wydner and Hoffman 1968). Due to these factors, there is a good deal of debate concerning the importance of community air pollution in the etiology of lung cancer.

Exposure Environment	Concentration ng/m^3	Effects
British gas works: Maximum levels above horizontal retort houses	200,000	Gas production workers had a 69% excess of lung cancer mortality and a 300% excess for bladder cancer mortality relative to workers with minimal exposure
Coal tar pitch working areas	75,000	Roofers employed 20 years or more had a 59% excess lung cancer mortality and excess mortality from other cancers relative to U.S. male population
Highest measured levels at topside of coke oven	42,700	Full topside workers employed 5 years or more had a 900% excess lung cancer mortality compared to all steelworkers
Average concentration topside of coke ovens	18,000	See effects at 42,700 ng/m^3
Roof tarring area	14,000	See effects at 75,000 ng/m^3
Average concentration side and bench areas of coke ovens	7,000	Side oven workers employed 5 years or more had a 46% excess lung cancer mortality compared to all steelworkers. Kidney cancer was elevated 469% for those working side oven and a mix of side and topside
Average concentration in British gas works	3,000	See effects at 200,000 ng/m^3
Ambient air equivalent of average mask measurements in roofing operations	2,000[a]	See effects at 75,000 ng/m^3
Lowest measured levels at topside of coke ovens	1,200	See effects at 42,700 ng/m^3
Great Britain industrial city, Winter 1955	170	Males living in urban areas tend to have about 100% excess lung cancer rates compared to rural residents
U.S. industrial city, Winter 1958	74	See effects at 170 ng/m^3
Ambient air equivalent of 1 1/2 packs of cigarettes per day	50[b]	Male smokers aged 35–84 have a 1400% excess lung cancer mortality compared to nonsmokers
Downtown Los Angeles, 1952–1953	31	Lung cancer mortality in males elevated 40% in area of highest pollution compared to the rate for the rest of L. A. county
Average for 100 U.S. cities, 1958	6	See effects at 170 ng/m^3
Median of average annual concentration at U.S. urban sites, 1966	3.2	See effects at 170 ng/m^3
Median of average annual concentrations at U.S. urban sites, 1975	0.5	See effects at 170 ng/m^3

[a] Assumes a breathing rate of 8 m^3 per 7 hour working day

[b] Assumes a breathing rate of 15 m^3 per 24 hours and 25 ng BaP per cigarette

Fig. 4.7. Levels and Effects of Benzo(a)pyrene (BaP)

In several studies, urban and rural populations were compared, and an attempt was made to attribute differences in lung cancer rates to the air pollution in urban areas. These studies tend to show, in males, a fairly consistent excess (about two-fold) in urban lung cancer rates over those found in the corresponding rural areas (Stocks and Campbell 1955; Manos and Fisher 1959; Levin et al. 1960; Haenzel et al. 1962). Both the Haenzel et al. and Stocks and Campbell studies included controls for smoking, but the conclusions regarding the interaction between smoking and residence were different for the two studies. Stocks and Campbell found that the urban-to-rural ratio of lung cancer mortality rates was greatest among nonsmokers and decreased to near unity in heavy smokers. Conversely, Haenszel et al. reported no differences in lung cancer mortality among nonsmokers in urban and rural areas and suggested that residence and smoking combine synergistically to affect lung cancer rates.

While urban dwellers seem to be at greater risk of lung cancer, there is no general consensus at the present time that these differences are due to air pollution. Higgins (1977) concludes that "while an urban effect is undoubted, it is still not certain that it is due to carcinogenic pollutants." Studies showing no consistent relationship between air pollution and lung cancer within the urban environment tend to support this contention. Winklestein and Kantor (1969), investigating mortality in Buffalo, and Hagstrom et al. (1967), investigating mortality in Nashville, found no association between lung cancer and particulate air pollution. However, in both studies a positive association between stomach cancer and particulate air pollution was found.

In a second type of community study, efforts have been made to correlate cancer mortality rates with some index of air pollution. Menck et al. (1974) found elevated lung cancer mortality in males living in three contiguous industrial areas of south-central Los Angeles County. The mortality rate in these three areas was 40% above the rate for the rest of Los Angeles County. BaP measurements revealed the highest pollution levels at the center of the three study areas (3.5 ng/m^3 in 1971-1972). These relatively recent measurements are assumed to be much lower than historic levels of 31 ng/m^3, the average concentration in downtown Los Angeles in 1952-53 (Gordon and Bryan 1973). In a followup study, Henderson et al. (1975) confirmed the elevated lung cancer rate for south-central Los Angeles County and reported that the excess risk was present across social classes and occupational groups.

Using county-level mortality rates, Blot et al. (1977) compared the cancer experience of those counties where the petroleum industry is most heavily concentrated to that of control counties without petroleum plants. Rates in the exposed counties were significantly elevated for cancers of the lung, nasal cavity and sinuses, and stomach. On the other hand, Higgins (1977) found no association between BaP levels or particulate matter and lung cancer mortality rates in 50 U.S. Standard Metropolitan Statistical Areas.

Carnow and Meier (1973) used a regression model to relate state-level lung cancer mortality rates to BaP concentrations while controlling for cigarette consumption. BaP concentrations used in the model were weighted estimates based on available measurements and the urban fraction of each state. The authors report that lung-cancer death rates for white males increased by 5%, with a 1 ng/m^3 increase in average BaP concentration, where BaP is used as a surrogate variable for all forms of general air pollution.

A final source of evidence on POM-associated health effects comes from the extensive literature on smoking. It is well established that lung cancer mortality rates increase with the amount of cigarette smoking (Hammond 1975), with roughly a 14-fold excess exhibited by those who smoke one to two packs a day. Less fully understood is the actual compound or mix of compounds that accounts for this large excess.

The smoker of one unfiltered cigarette inhales about 25 ng BaP (National Research Council 1972b) or 500 ng BaP/pack. Assuming a breathing rate of 15 m^3/day, an ambient air concentration of roughly 50 ng/m^3 BaP would deliver, in one day, the equivalent BaP of 1-1/2 packs of cigarettes (Handy and Schindler 1976). This simplified calculation illustrates the difficulty in interpreting the health effects of POM based on available data. The 50 ng/m^3 BaP is within the range of ambient values for an industrial urban area and far below the concentrations in many occupational settings discussed above (see Fig. 4.7). Yet, in neither community nor occupational studies has a relationship between exposure and disease been discovered that approaches the magnitude and consistency of the smoking and lung cancer association. Obviously, there are important factors operating that are not yet well understood.

4.2.2.4 Subcellular and Cellular Effects in Laboratory Animals

The enormous number and complexity of chemical structures of products derived from coal combustion and other processes such as water leaching of coal ash piles can, in turn, produce a wide variety of biological effects. Some of the PNAs are toxic to various types of cells upon exposure. Others are not toxic until they are acted upon by intracellular enzyme systems, which convert them into various metabolites, some of which can be toxic. Many of these compounds are also mutagenic when tested in appropriate mutagenesis assay systems, although some of the compounds

must again be enzymatically altered in order to induce mutagenesis. Some of these PNAs (or their metabolites) also can cause certain morphological and biochemical alterations in cell cultures exposed to them, and some of these altered cells can eventually become tumorigenic when injected into the appropriate host animal. This process is known as transformation. Indeed, a major concern of much biological research is the potential carcinogenicity of at least some of the coal-associated chemicals.

The subcellular perturbations produced by coal-associated chemicals cover a wide spectrum of processes; covalent binding of the PNA to various macromolecules and changes in certain enzyme activities is briefly addressed here. Much attention has been focused on covalent binding of some of the PNA to macromolecules such as DNA, RNA, and protein. Particular emphasis has been placed on DNA binding, because of the importance of DNA as the genetic material of the cell. The PNA must be metabolically activated by appropriate enzyme systems for this binding to occur. Although some correlation between tumorigenicity of certain PNA in vivo and the amount bound to a given amount of DNA could be demonstrated, the correlation holds for only certain compounds (Santodonato et al. 1979). Furthermore, other work has shown some correlation between in vivo tumorigenicity and amount of binding to certain proteins (Santodonato et al. 1979).

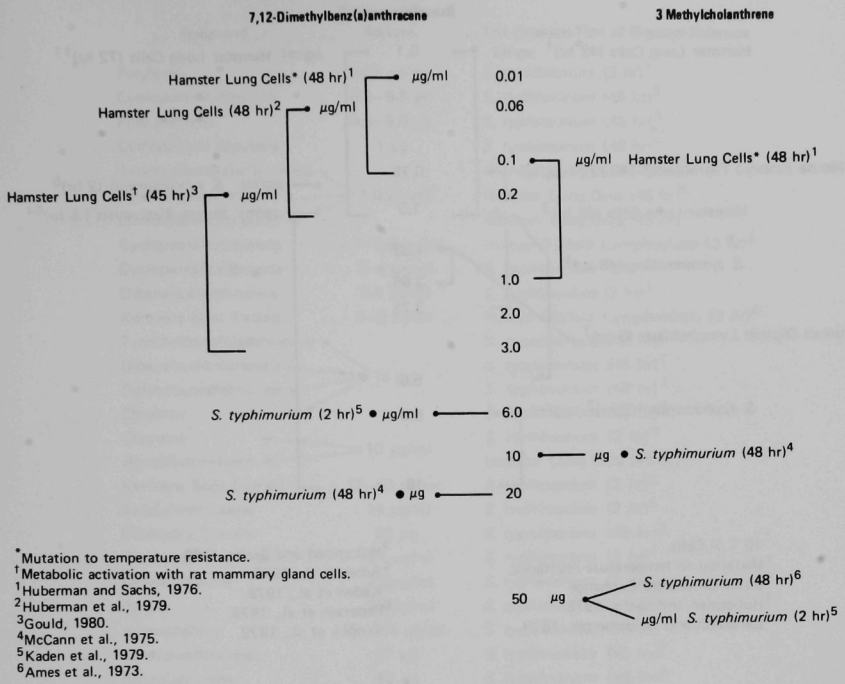
The PNA produce significant effects upon enzymatic systems in cells exposed to them. One of the most important is induction of increased enzymatic activity of the enzyme aryl hydrocarbon hydroxylase (AHH) (a fraction of the larger group of enzymes called the mixed-function oxygenases). This enzyme is the protein responsible for the initial steps in metabolizing PNA, and in concert with other enzymes, its biological function is to render lipid-soluble PNA excretable in aqueous fluids. This enzyme, in the process of oxygenating the PNA, also can produce metabolites that will interact with cellular macromolecules; some of these metabolites are suspected of being the actual cellular carcinogen, rather than the parent compound (Santodonato et al. 1979). There does not seem to be a one-to-one correspondence between a particular PNA's in vivo animal tumorigenicity and its ability to increase AHH activity, since some rather potent AHH inducers are rather weak animal carcinogens (Santodonato et al. 1979; Walsh et al. 1978, Appendix D).

There is evidence that other coal-associated chemicals (e.g., cadmium) also can induce mixed-function oxygenases (Means and Schnell 1979). Thus, synergistic effects of certain trace elements and PNA may result in increased production of carcinogenic metabolites in cells exposed to complex mixtures. The problem of synergistic and antagonistic effects of complex mixtures cannot be adequately addressed at this time due to lack of basic biological information.

Some of the cellular effects of some coal-associated PNA are shown in Figures 4.8 through 4.19. Although other effects could be listed, a general idea of overall cellular effects can be gained from considering the toxicity of the PNA (or its metabolites) to in vitro cellular systems, the ability of the PNA to act as a mutagen in appropriate systems, and ability of the PNA to induce in vitro transformation. The PNA must be metabolized to become cytotoxic in vitro, while certain other chemicals such as NO_2, SO_2, and the trace metals are directly cytotoxic (Walsh et al. 1978). The degree of toxicity of a given chemical varies with different cell lines. Thus, the cytotoxicity evaluations for PNA in Figure 4.16 are all relative to benzo(a)pyrene, which was called strongly toxic (Walsh et al. 1978, Appendix D). SO_2 and NO_2 were evaluated as strongly toxic compared to CO (Walsh et al. 1978, Appendix D). Cytotoxic estimations for the four trace metals were made relative to cadmium.

Mutagenicity of various coal-associated PNA is indicated in Figures 4.8 through 4.12 as positive or negative. The summary figure of results for PNA and other organic compounds refers only to assays done in the Ames system (McCann et al. 1975) or using the S. typhimurium assay of Kaden et al. (1979). No attempt was made to quantify mutagenicity, since the degree of mutagenicity of the various PNA is very dependent upon the metabolic activating system used in the test. Kaden et al. (1979) have found that mutagenicity of individual compounds of a synthetic soot could account for mutagenicity of the whole soot extract in a simple additive fashion. This does not mean, however, that synergistic mutagenic effects are not to be expected in complex mixtures of coal-associated chemicals. The mutagenic potentials of cadmium, manganese, nickel, and lead have been estimated only to the extent of misincorporation in in vitro polydeoxyribonucleotide synthesis experiments. In contrast, arsenic and chromium have induced mutations in rather standard mutagenicity assays.

The ability of various PNA and other coal-associated chemicals to produce morphological transformatism in various transformable mammalian cell lines also is indicated in Figures 4.17 through 4.19. Again, no attempt was made to quantitate this parameter, because of inherent variability in the transformable fraction of cells in different cell lines. If a report could be found of transformation in at least one cell line, the chemical is listed as positive. The absolute relationship between in vitro transformation and in vivo carcinogenesis is at present unclear, but it has been shown that a number of animal carcinogens also are capable of causing in vitro transformation.



*Mutation to temperature resistance.
†Metabolic activation with rat mammary gland cells.
[1]Huberman and Sachs, 1976.
[2]Huberman et al., 1979.
[3]Gould, 1980.
[4]McCann et al., 1975.
[5]Kaden et al., 1979.
[6]Ames et al., 1973.

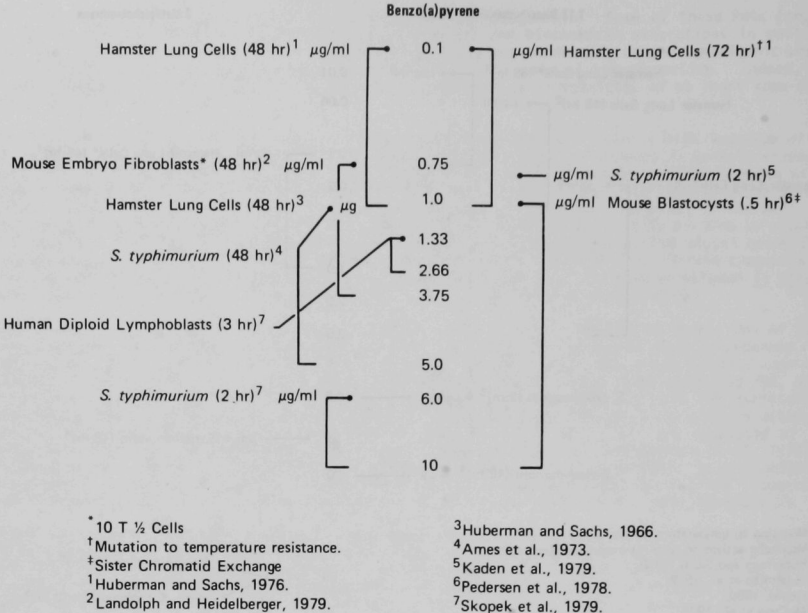
Fig. 4.8. Genetic Effects--Mutation: 7,12-Dimethylbenz(a)anthracene and 3 Methylcholanthrene

Genetic Effects

The abilities of various polynuclear aromatic hydrocarbons (PNAs) and certain other compounds to cause detectable mutations in a variety of in vitro systems is indicated in the accompanying tables (Figures 4.8 through 4.12). Data on benzo(a)pyrene (BaP), 7,12-dimethylbenz(a)anthracene (7,12-DMBA) and 3-methylcholanthrene (MCA) are tabulated separately from other PNA because these three compounds are often used as "standard" or "surrogate" chemicals for the whole class of PNA, and hence, more information is available for these compounds.

Several points should be made with respect to the data tabulated. First, most or all of these compounds require metabolic activation in order to be mutagenic (that is, they must be modified to a more chemically reactive state by certain enzymes), and some of the common cell lines used in mutagenic assays do not have the necessary enzymatic machinery to accomplish this. Examples of such systems would be the Ames test, utilizing S. typhimurium, and the hamster lung cells (V79) utilized by Huberman and others. The Ames system commonly utilizes rat liver microsomes, and these microsomes usually are obtained from rats treated with various monooxygenase inducers, such as Arochlor 1254, a polychlorinated organic compound. Investigators using the V79 cell assay have used a variety of activation systems including "feeder" layers of secondary hamster embryo cells, rat liver hepatocytes, fibroblasts, and rat mammary gland cells. These "feeder" layer cells are plated in the same dish as the V79 cells, and PNAs must first be metabolized in these cells, and then the metabolites must pass out of the metabolizing cells, and somehow (through unknown mechanisms) pass through the membrane of the V79 cells (without reacting with membrane components) and react with intracellular macromolecules. Where not specifically indicated, the activation system used for V79 cells was hamster embryo cells or hamster fibroblasts.

In the case of systems employing human diploid lymphoblasts, or S. typhimurium exposed for 2 hours (Skopek et al. 1979; Krishnan et al. 1979), chemicals were activated with rat liver



*10 T ½ Cells
†Mutation to temperature resistance.
‡Sister Chromatid Exchange
[1]Huberman and Sachs, 1976.
[2]Landolph and Heidelberger, 1979.
[3]Huberman and Sachs, 1966.
[4]Ames et al., 1973.
[5]Kaden et al., 1979.
[6]Pedersen et al., 1978.
[7]Skopek et al., 1979.

Fig. 4.9. Genetic Effects--Mutation: Benzo(a)pyrene

microsomes from rats treated with Arochlor 1254. Mouse embryo fibroblast experiments did not utilize a separate metabolic activation system.

The use of various activation systems makes comparisons of the genotoxic activities of various chemicals extremely difficult. This is because no real concept of "dose" of toxic agent can be developed, and hence, comparisons of chemicals on the basis of equivalent dose cannot be made. The concept of dose = concentration of toxicant x time of exposure cannot be applied to these complex in vitro systems, because of inability to know precisely (1) the concentration of toxicant the target cells are exposed to and (2) the time of such exposure. Because these chemicals must be metabolically activated, the concentration of chemical initially put in the cell culture medium may have little bearing on the amount of toxic metabolite actually involved in genetic damage. In fact, this (or these) genotoxic metabolite(s) probably will be a variable percentage of the total metabolism, the variability being due to the fact that different metabolic pathways have different overall rates. The estimation of effective concentration is further confounded by the fact that most metabolic activation systems are external to the target cells, so that diffusion of genotoxic metabolites must be involved, and these "active" metabolites probably are inactivated to varying degrees by medium components, cell membranes, etc. In the case of many organic compounds (particularly the PNA), the situation is further complicated because of limited solubility (or insolubility) of the chemical in aqueous solution. The compound usually is introduced by dissolving in acetone, dimethyl sulfoxide, or some other solvent, and then adding a small amount of this solution to the growth medium. Quantification of effective chemical concentration is clearly dependent on many factors whose individual contributions to overall variability is unknown.

In the same manner, it is impractical to attempt to estimate actual exposure times from experimental length of chemical exposure recorded, since the rate at which a given metabolic activation system produces genotoxic metabolites probably is quite variable, and certainly is incomparable between different activation systems.

Compound	Concentration or Amount	Test Organism-Time of Exposure-Reference
Perylene	0.25 μg/ml	*S. typhimurium* (2 hr)[1]
Cyclopenta(c,d)pyrene	0.8–6.8 μg	*S. typhimurium* (48 hr)[2]
Phenanthrene	0.9–9.0 μg	*S. typhimurium* (48 hr)[3]
Cyclopenta(c,d)pyrene	1 μg	*S. typhimurium* (48 hr)[4]
7-methylbenz(a)anthracene	1.0 μg/ml	Hamster Lung Cells (48 hr)[5]
Dibenz(a,c)anthracene		Hamster Lung Cells (48 hr)[5]
Dibenz(a,h)anthracene		Hamster Lung Cells (48 hr)[5]
Cyclopenta(c,d)pyrene	1.3–2.6 μg/ml	Human Diploid Lymphoblasts (3 hr)[5]
Cyclopenta(c,d)pyrene	3–6 μg/ml	*S. typhimurium* (2 hr)[6]
Dibenz(a,c)anthracene	3.6 μg/ml	*S. typhimurium* (2 hr)[1]
Kerosene Soot Extract	4–8 μg/ml	Human Diploid Lymphoblasts (3 hr)[6]
7-methylbenz(a)anthracene	10 μg	*S. typhimurium* (48 hr)[7]
Dibenz(a,c)anthracene		*S. typhimurium* (48 hr)[7]
Dibenz(a,h)anthracene		*S. typhimurium* (48 hr)[7]
Chrysene		*S. typhimurium* (48 hr)[7]
Chrysene	10 μg/ml	*S. typhimurium* (2 hr)[1]
Benz(a)anthracene		Hamster Lung Cells (48 hr)[8]
Kerosene Soot Extract	12–50 μg/ml	*S. typhimurium* (2 hr)[6]
Benz(a)anthracene	14 μg/ml	*S. typhimurium* (2 hr)[1]
Dibenzo(a,i)pyrene	20 μg	*S. typhimurium* (48 hr)[7]
Dibenz(a,h)anthracene	21 μg/ml	*S. typhimurium* (2 hr)[1]
Benzo(e)pyrene	23 μg/ml	*S. typhimurium* (2 hr)[1]
Pyrene	28 μg/ml	*S. typhimurium* (2 hr)[1]
Acenaphthene	46–154 μg/ml	*S. typhimurium* (2 hr)[9]
Benz(a)anthracene	57 μg	*S. typhimurium* (48 hr)[7]
Benzo(e)pyrene	60 μg	*S. typhimurium* (48 hr)[7]
5-Cyanoacenaphthene	90–500 μg/ml	*S. typhimurium* (2 hr)[9]
1-Cyanoacenaphthylene	250–500 μg/ml	*S. typhimurium* (2 hr)[9]
5-Cyanoacenaphthylene		*S. typhimurium* (2 hr)[9]

[1]Kaden et al., 1979.
[2]Wood et al., 1980.
[3]Wood et al., 1979.
[4]Eisenstadt and Gold, 1978.
[5]Huberman and Sachs, 1976.
[6]Skopek et al., 1979.
[7]McCann et al., 1975.
[8]Slaga et al., 1978.
[9]Krishnan et al., 1979.

Fig. 4.10. Genetic Effects--Mutation: Miscellaneous PNAs

For these reasons, comparisons of mutagenic potencies on the basis of chemical dose between different mutation systems probably are meaningless. The whole matter of relative mutagenicity is further complicated by the fact that the end-point of the mutation assay (the actual detection of an altered gene product) varies from system to system.

In the case of hamster lung cells, the two most common mutation endpoints are mutations affecting the genetic loci controlling the hypoxanthine-guanine phosphoribosyl transferase (HGPRT) enzyme or the ouabain-sensitive adenosine triphosphatase (ATPase) enzyme. In a few instances, mutations to temperature resistance are used as the end point in this cell line. In the case of S. typhimurium assays involving the Ames system, mutation involves alteration in the genes controlling the histidine biosynthetic pathway. The system involving 2-hr exposure of S. typhimurium to the chemical of interest uses the HGPRT locus as a mutagenesis marker, as does the human diploid lymphoblast system. The mouse embryo fibroblast system utilizes the ouabain-sensitive APTase locus as a mutagenesis marker. Whether or not the amount of genetic material (as a proportion of the total DNA content) being monitored as the target for mutagenic events is at all comparable in the different systems is a moot point.

Compound	Concentration or Amount	Test Organism Time of Exposure-Reference
Dimethylnitrosamine	0.3–300 μg/ml	Hamster Lung Cells* (18 hr)[1]
Diethylnitrosamine	0.4–400 μg/ml	Hamster Lung Cells* (18 hr)[1]
2-Naphthylamine	10 μg	*S. typhimurium* (48 hr)[2]
2-Naphthylamine	100 μg	*S. typhimurium* (48 hr)[3]
Diethylnitrosamine	250–2500 μg/ml	Hamster Lung Cells* (48 hr)[4]
Dimethylnitrosamine	300–900 μg/ml	Hamster Lung Cells* (48 hr)[4]
Diethylnitrosamine	4080 μg	*S. typhimurium* (48 hr)[2]
Dimethylnitrosamine	4440 μg	*S. typhimurium* (48 hr)[2]

*Rat liver hepatocytes used for metabolic activation.
[1]Jones and Huberman, 1980.
[2]McCann et al., 1975.
[3]Ames et al., 1973.
[4]Langenbach et al., 1978.

Fig. 4.11. Genetic Effects--Mutation: Other Chemicals

In most cases, the concentrations of various chemicals which produce significant levels of mutation are indicated in the tables by bars encompassing concentration ranges. The lower end of these concentration ranges is selected as the minimum concentration, which still produces a significant mutation frequency. This lower limit is not particularly well defined. In the case of some Ames system assays (S. typhimurium, 48-hr exposure), the single concentration of chemical designated in many cases was taken from a linear dose-response curve for that chemical and does not represent the lowest concentration of that chemical which had mutagenic activity (see McCann et al. 1975). Nevertheless, this single concentration can still provide a relative ordering of mutagenic potency of chemicals, which probably is the most useful way of utilizing these data. A further problem in comparing the Ames system with any other mutagenicity assays is that the concentration of chemical in the Ames system is expressed as μg/plate. Dosimetric comparisons between different systems thus are very difficult. In the case of the data of Kaden et al. (1979) for S. typhimurium (2-hr exposure), the single concentration given is the lowest concentration at which mutagenic activity was detected in this system, so that relative comparisons of chemicals assayed by this procedure can be made easily. The time of exposure for each assay procedure is indicated in parentheses in the appropriate figures.

For all of these reasons, comparisons of chemical potencies using different mutagenesis systems are not very meaningful. A summary figure (Fig. 4.12) has been prepared in which a number of chemicals are compared in two systems: (1) the Ames system and (2) mutagenesis in S. typhimurium using the HGPRT enzyme locus as a mutation marker. These represent only relative potencies and any attempt to interpret these relative potencies as having quantitative meaning should be done with extreme caution. For example, it might be said that benzo(a)pyrene is a more potent mutagen than benzo(e)pyrene in the Ames system; yet it is probably stretching the limitations of the system to say that benzo(a)pyrene is a more potent mutagen than 3-methylcholanthrene. Only orders-of-magnitude differences in mutagenic response to chemicals have much meaning in applying these data to relative mutagenic potencies.

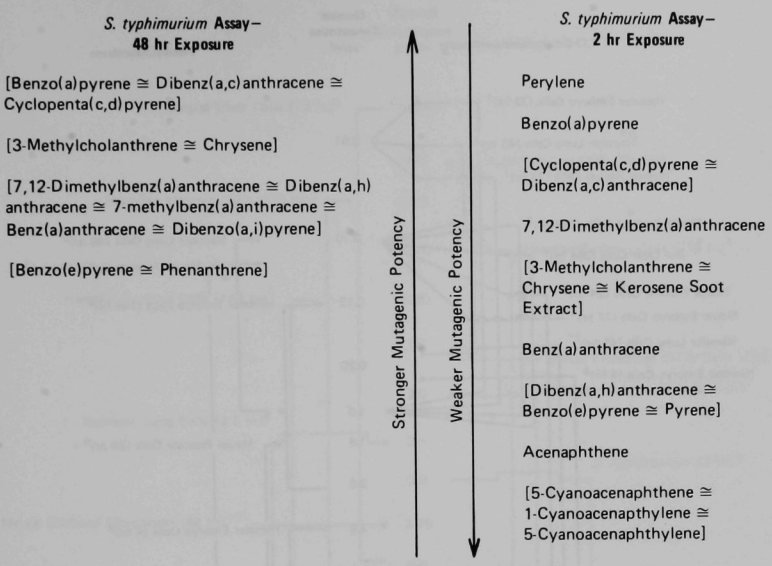


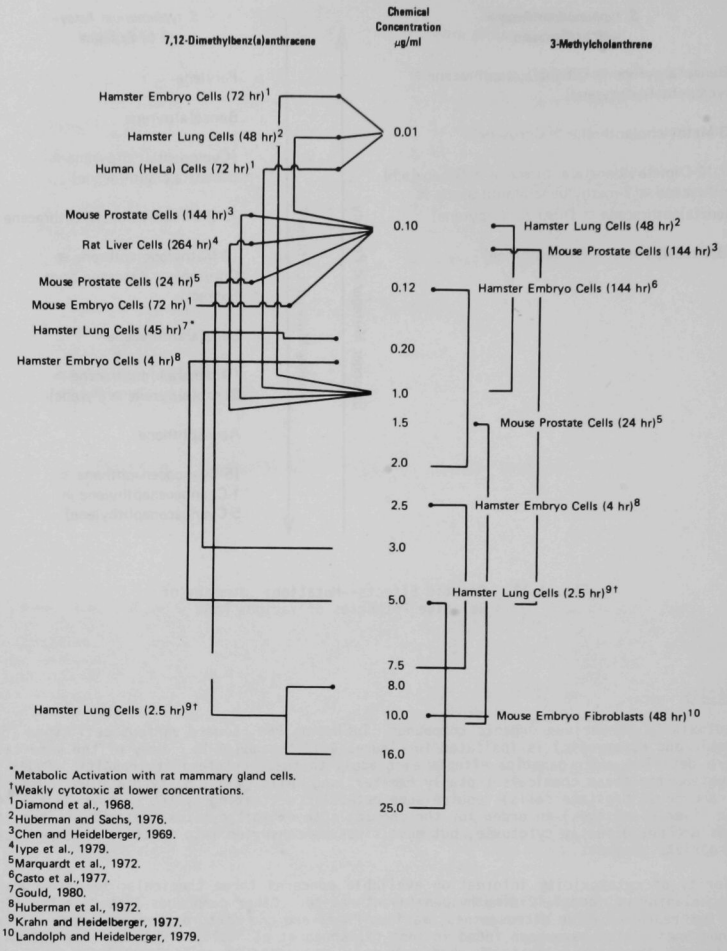
Fig. 4.12. Genetic Effects--Mutation: Summary of Relative Potencies of Various PNAs

Cytotoxicity

The cytotoxicity of various organic compounds, including PNA, toward various cell types (both prokaryotic and eukaryotic) is indicated in Figures 4.13 through 4.15. Many of the same caveats that were detailed under genetics effects also apply to these cytotoxicity results. Cells that cannot metabolize these chemicals (notably hamster lung cells [V79], S. typhimurium, human lymphoblasts, and mouse prostate cells) require some metabolic activating system (either a cell "feeder" layer or liver microsomes) in order for the chemicals to exhibit cytotoxic effects. Thus, these compounds are not directly cytotoxic, but must first be converted into reactive intermediates by the appropriate enzymes.

The majority of cytotoxicity information available concerns three chemicals: benzo(a)pyrene, 3-methylcholanthrene, and 7,12-dimethylbenz(a)anthracene. Other compounds listed in the figures include two representative nitrosamines, various PNAs, and one mixture (kerosene soot extract). The cyanoarenes listed have been found in soot (Krishnan et al. 1979). Cytotoxicity information in the figures has been selectively chosen in this sense; the lower limit at which a chemical shows a cytotoxic effect is selected on the basis of a reduction to at least 75-80% relative cell survival (cell survival of cells exposed to a given concentration of a chemical ÷ cell survival of cells exposed to solvent x 100). This survival limit is chosen because it is rather difficult to statistically distinguish survival data in this low cell-killing region (i.e., 80-90% survival), and experiments must be specifically targeted to obtain such information reliably. In most experiments from which this review was compiled, a broad range of concentrations were tested, with no specific attempt to elucidate the shape of the survival curve at low cell killing.

As pointed out in the analysis of genetic effects, it is extremely difficult to make comparisons of the relative toxic efficiency of these various compounds. First, the methods of metabolic activation vary, from using cells that can directly metabolize chemicals as target cells, to "feeder" cells that must first metabolize and then "pass on" reactive metabolites to target cells, to microsomal enzyme preparations that generate reactive metabolites in solution, which must then reach target cells through the medium of this solution. Comparisons of dosimetry between differing metabolic activating systems are little more than guesses.



*Metabolic Activation with rat mammary gland cells.
†Weakly cytotoxic at lower concentrations.
[1]Diamond et al., 1968.
[2]Huberman and Sachs, 1976.
[3]Chen and Heidelberger, 1969.
[4]Iype et al., 1979.
[5]Marquardt et al., 1972.
[6]Casto et al.,1977.
[7]Gould, 1980.
[8]Huberman et al., 1972.
[9]Krahn and Heidelberger, 1977.
[10]Landolph and Heidelberger, 1979.

Fig. 4.13. Cytotoxicity of PNAs

Second, there may be differential resistance of various cell lines to the toxic effect of chemicals. This is not envisioned to be a severe barrier, simply because it is likely that the multiple events that may lead to cell death are probably quite similar for different mammalian cells, since they probably occur in the more fundamental metabolic pathways common to most cells. Comparisons of cytotoxic effect between eukaryotic cells and prokaryotic (S. typhimurium specifically) cells could be subject to this criticism.

Third, there are potentially several ways of assaying cytotoxic activity, and comparisons between results from different methods are very difficult to make. Almost all the experiments cited in this document have measured cell survival by cloning efficiency, i.e., the ability of a single cell to form a micro- or macroscopic colony which is then counted as evidence that the initial progenitor cell was reproductively viable. This probably is a more rigorous criterion of cell

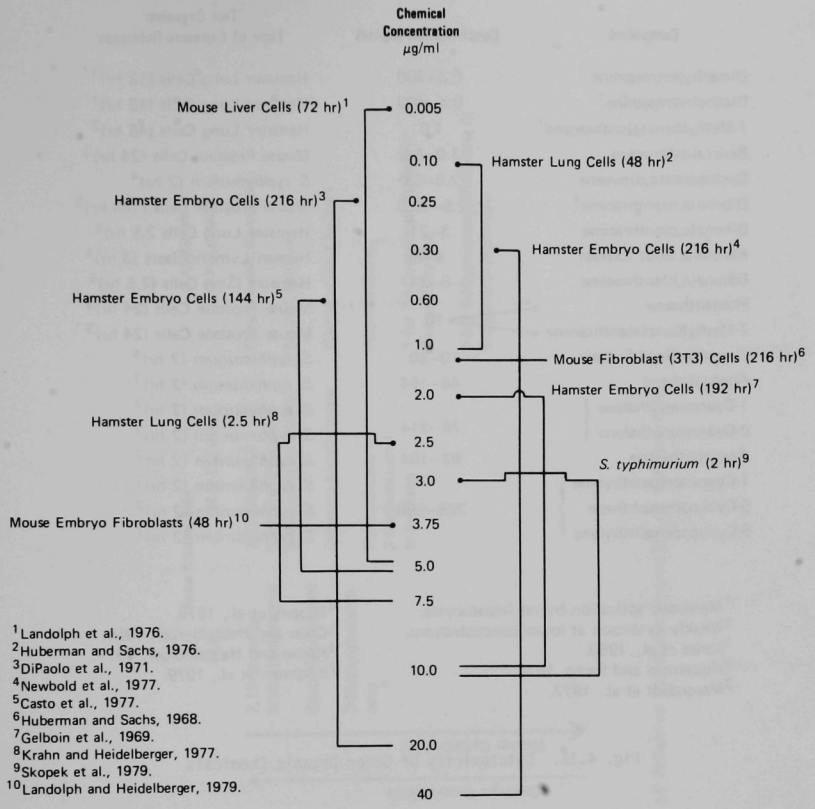


Fig. 4.14. Cytotoxicity of Benzo(a)pyrene

viability than are trypan blue or other dye-exclusion assays. Even in reproductive survival assays, methodological procedures (such as whether cells are seeded at the very low density necessary for subsequent accurate colony counting before or after exposure to the chemicals) may significantly differ, giving rise to great uncertainty in evaluating comparisons.

Fourth, the times of exposure to the chemical are very variable, and there seems to be no very good way to correct for this variability. This exposure-time variability is at least partially related to the differences in assay methodology previously discussed. An initial concept that might be used to try to "standardize" the effect of various exposure times would be the concept of "dose", that is dose = concentration of chemical x time of exposure. For this calculation to have any real accuracy, it is necessary to know concentrations of chemical actually impinging the cell, and, for reasons already discussed, this parameter is very poorly defined in these systems. Thus "doses" to cells are, at best, only gross estimates.

Some representative calculations to test the validity of this "dose" concept for various chemicals are indicated below. These were calculated simply by multiplying the lowest cytotoxic concentration recorded times the exposure time. These comparisons were only made within the constraints of a single cell culture system, such as hamster lung cells (V79), etc. Minimal cytotoxic "doses" (units of μg-hr/mL) for benzo(a)pyrene were: hamster lung cells-4.8, 6.3; hamster embryo cells-5.9, 8.6, and 384. Similar calculations for 7,12-dimethylbenz(a)anthracene and for 3-methylcholanthrene yielded respectively: hamster lung cells-0.5, 0.9, and 20.0 for DMBA; 0.5, 12.5 for

Compound	Concentration (μg/ml)	Test Organism Time of Exposure-Reference
Dimethylnitrosamine	0.3–300	Hamster Lung Cells (18 hr)[1]*
Diethylnitrosamine†	0.4–400	Hamster Lung Cells (18 hr)[1]*
7-Methylbenz(a)anthracene†	1.0	Hamster Lung Cells (48 hr)[2]
Benz(a)anthracene	1.0–5.0	Mouse Prostate Cells (24 hr)[3]
Cyclopenta(c,d)pyrene	2.0–6.0	*S. typhimurium* (2 hr)[4]
Dibenz(a,h)anthracene†	2.5–10.0	Mouse Prostate Cells (144 hr)[5]
Dibenz(a,c)anthracene	3–21	Hamster Lung Cells 2.5 hr)[6]
Kerosene Soot Extract	4–8	Human Lymphoblasts (3 hr)[4]
Dibenz(a,h)anthracene	6–24	Hamster Lung Cells (2.5 hr)[6]
Phenanthrene	10	Mouse Prostate Cells (24 hr)[3]
7-Methylbenz(a)anthracene		Mouse Prostate Cells (24 hr)[3]
Kerosene Soot Extract†	10–50	*S. typhimurium* (2 hr)[4]
Acenaphthene	46–154	*S. typhimurium* (2 hr)[7]
1-Cyanonaphthalene	76–214	*S. typhimurium* (2 hr)[7]
2-Cyanonaphthalene		*S. typhimurium* (2 hr)[7]
Acenaphthylene	92–184	*S. typhimurium* (2 hr)[7]
1-Cyanoacenaphthylene	268–500	*S. typhimurium* (2 hr)[7]
5-Cyanoacenaphthene		*S. typhimurium* (2 hr)[7]
5-Cyanoacenaphthylene		*S. typhimurium* (2 hr)[7]

*Metabolic activation by rat hepatocytes.
†Weakly cytotoxic at lower concentrations.
[1]Jones et al., 1980.
[2]Huberman and Sachs, 1976.
[3]Marquardt et al., 1972.
[4]Skopek et al., 1979.
[5]Chen and Heidelberger, 1969.
[6]Krahn and Heidelberger, 1977.
[7]Krishnan et al., 1979.

Fig. 4.15. Cytotoxicity of Other Organic Chemicals

MCA; hamster embryo cells-0.7, 0.8 for DMBA; 1.7, 10 for MCA. It can be seen that in most cases a fair degree of agreement (within an order of magnitude) exists for these calculated doses. This suggests that the problem of varying exposure times may be at least partially solved by making such simple dose calculations. The caution must be reiterated, however, that such dosage corrections can be applied meaningfully only to chemical comparisons for a single cell line, because of tremendous differences in growth cycles (and control of same) and response to chemicals among various cells.

A comparison of the relative cytotoxicities of various chemicals is presented in Figure 4.16; these comparisons are made strictly for a single-cell culture system and for a given exposure time. In general (subject to the caveats listed above), 7,12-dimethylbenz(a)anthracene seems to be the most cytotoxic PNA, followed closely by 3-methylcholanthrene, benzo(a) pyrene, and probably dimethyl- and diethylnitrosamine. PNA is not so strongly toxic, but of moderately strong toxicity are benz(a)anthracene, 7-methylbenz(a)anthracene, cyclopenta(c,d)pyrene, dibenz(a,h)anthracene, dibenz(a,c) anthracene, kerosene soot extract, and phenanthrene. More weakly cytotoxic agents would include the cyanoarenes.

The cytotoxic activity of various chemicals may be important not only from a standpoint of indicating the toxic nature of various chemical species, but also in providing a measure of cancer-promoting activity of various chemicals. Some means of estimating promotional efficacy of various compounds is sorely needed, as this promotional aspect may be of more significance to the development of actual tumors than the initiating event that first generates a neoplastic cell. This concept of use of cytotoxicity data as a stimulus for proliferative-promotional cellular growth response is discussed more fully in Griffin et al. (1979) and Jones et al. (1980).

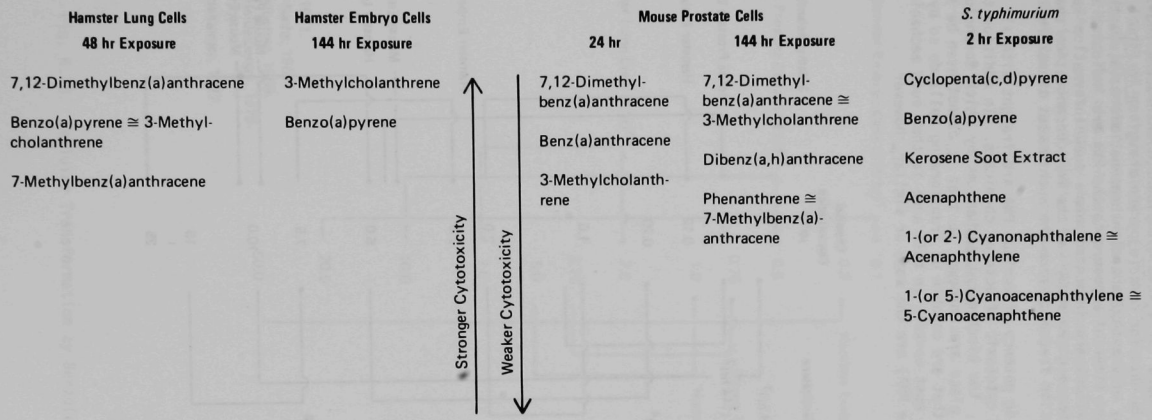
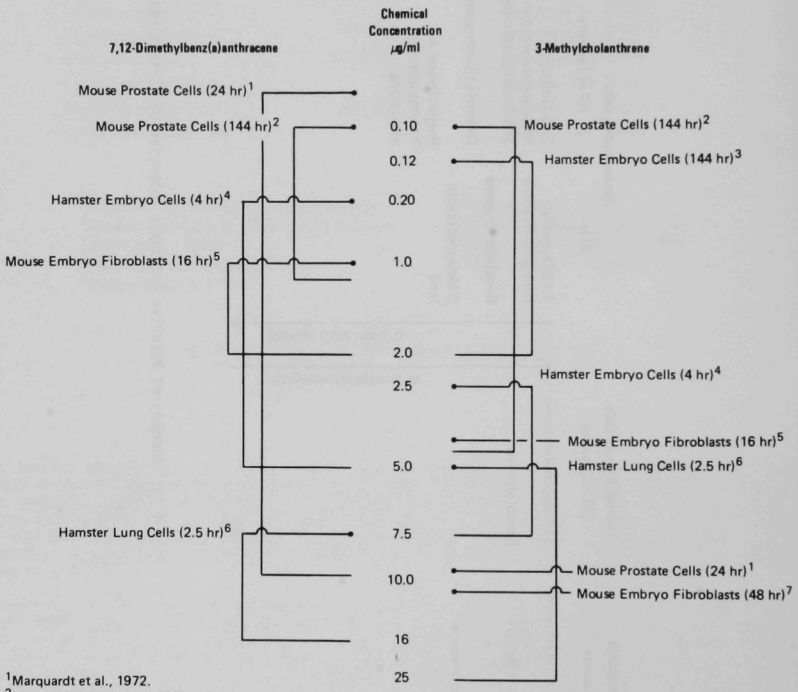


Fig. 4.16. Summary of Relative Cytotoxic Potencies of Various PNAs

Transformation

The ability of various PNA to produce a morphological transformation in various mammalian cells is indicated as a function of concentration of the chemical the cells are exposed to in Figures 4.17 through 4.19. Transformation data for 7,12-dimethylbenz(a)anthracene and 3-methylcholanthrene are given in Figure 4.17, while data for benzo(a)pyrene are given in Figure 4.18, and information about the transforming ability of 7-methylbenz(a)anthracene, dibenz(a,c)anthracene, and dibenz(a,h)-anthracene in Figure 4.19. The times of exposure to the PNA are indicated in parentheses in the figures. Ranges of concentrations are indicated where such information was available. The lowest concentration listed for a given study was the PNA concentration that provided a significantly different transformation frequency from the spontaneous transformation rate.

Comparisons of the transforming potency of various PNA are fraught with all the hazards mentioned previously for mutational and cytotoxic effects of chemicals. In addition to imprecise dosimetry, different solubilities, etc., the biochemical mechanisms by which morphological alterations (called transformation) take place are unknown, so that it cannot even be said with certainty that any two given chemicals that are capable of transforming cells do so by the same mechanism. The tacit assumption made by most workers in this area is that such mechanisms, for at least a given class of chemicals (like PNA) are the same or similar, however.



[1]Marquardt et al., 1972.
[2]Chen and Heidelberger, 1969.
[3]Casto et al., 1977.
[4]Huberman et al., 1972.
[5]Bertram, 1977.
[6]Krahn and Heidelberger, 1977.
[7]Landolph and Heidelberger, 1979.

Fig. 4.17. Cellular Transformation by PNAs

The underlying significance, from a biological viewpoint, of morphologically transformed cells produced in response to a chemical is not altogether clear. It is true that most such transformed cells give rise to fibrosarcomas when injected into suitably prepared (immuno-suppressed) host animals (Santodonato et al. 1979). Thus, morphological transformation has been suggested as a neoplastic transformation of cells occurring in vitro. Whatever the ultimate interpretation finally applied to in vitro transformation in the light of advances in mechanistic knowledge, in vitro transformation, at the present stage of knowledge, seems a reasonably good indicator of the ability of a chemical to function as a carcinogen. In this light, it is not very surprising that the PNA listed in the figures show cellular transformation ability, as most, if not all, of these compounds cause various forms of neoplastic-like growth in animals. Whether these results in animals can be extrapolated to humans is another question.

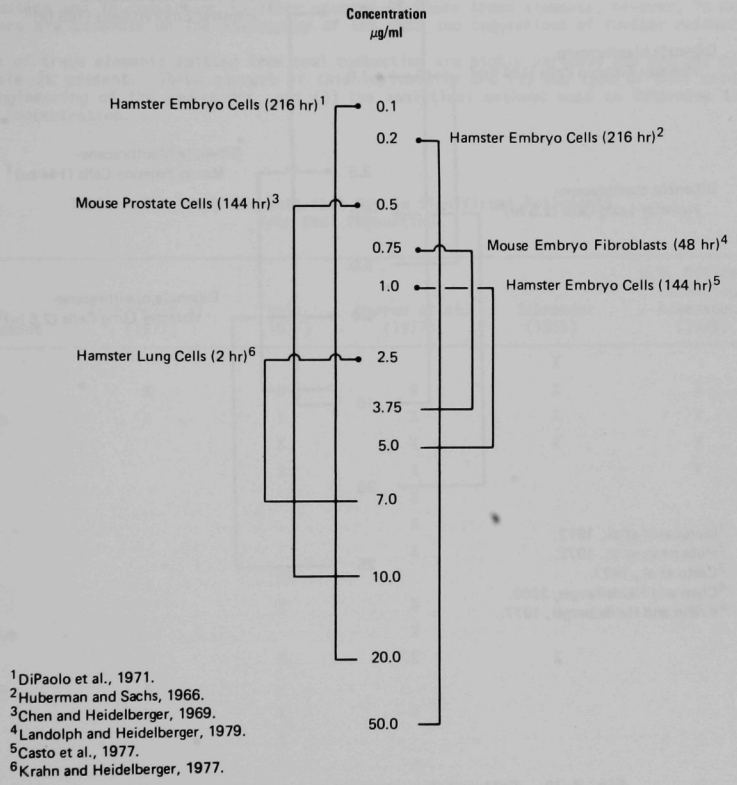


Fig. 4.18. Cellular Transformation by Benzo(a)pyrene

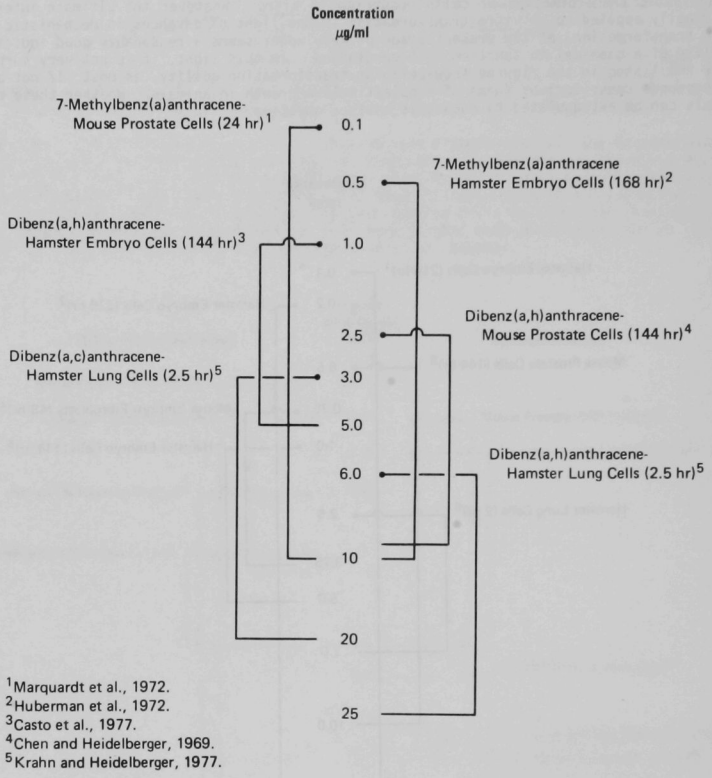


Fig. 4.19. Cellular Transformation by Miscellaneous PNAs

4.3 TRACE ELEMENTS

4.3.1 Introduction

Many trace elements have been identified as being of potential concern in relation to health effects, and coal combustion frequently has been cited as a source of atmospheric emissions of these elements. The elements identified in various studies are listed in Table 4.7. Using these studies as guides, the following elements have been singled out: Arsenic (As), beryllium (Be), cadmium (Cd), chromium (Cr), mercury (Hg), nickel (Ni), lead (Pb), selenium (Se), and thallium (Tl). Several assessments have been made as an attempt to determine the potential threat to human health from trace elements (Vaughn et al. 1975; Morrow et al. 1977; U.S. Dept. of Health, Education and Welfare 1978; Van Hook 1979; U.S. Office of Technology Assessment 1979). It is suggested in these reports that although there may be some potential for concern, trace element emissions will assume a minor role in comparison to other pollutants emitted by coal combustion and in comparison to other sources of these trace elements; however, in each report there are comments on the inadequacy of the data and suggestions of further research.

The amount of trace elements emitted from coal combustion are highly variable and are not adequately quantifiable at present. Three sources of this variability are (1) the type of coal used, (2) the engineering of the powerplant, and (3) the analytical methods used to determine the elemental concentration.

Table 4.7. Trace Elements that May Be Significant Pollutants from Coal Combustion

Trace Element	Friberg et al. (1977)	Heit (1977)	Morrow et al. (1977)	Schroeder (1965)	U.S. Office of Technology Assessment (1979)
Antimony				X	
Arsenic	X	X	X	X	X
Beryllium	X	X	X	X	X
Cadmium		X	X	X	X
Chromium		X	X		X
Cobalt			X		
Copper			X		
Fluorine			X		
Lead		X			
Mercury		X	X	X	X
Molybdenum			X		
Nickel		X	X	X	
Tellurium	X				
Thallium	X	X	X		
Tin		X			
Tungsten			X		
Selenium		X	X		X
Vanadium			X		
Zinc			X		

Trace elements in coal originate from weathering of uplands surrounding a coal-forming swamp. The amount and relative percentage of the incorporated trace element depends on the geology of the coal-forming basin and the physiochemistry of the element (Zubovic 1975). Most of the elements in coal are at concentrations near average crustal abundance; however, cadmium, boron, and selenium are enriched in coal by at least an order of magnitude, while fluorine, manganese, and phosphorus are depleted by at least an order of magnitude (Ruch et al. 1974). Concentrations may be quite variable in a given coal seam and may be even more variable in samples taken from different coal basins in different parts of the country. This variability is shown in Table 4.8. Considering this variability, care must be taken when averaging regional trace-element composition of coals (Magee et al. 1973; Dvorak et al. 1977). Vaughan et al. (1975) predicted maximum credible concentrations of trace elements in the air near a 1400-MWe coal-fired powerplant. Values for trace elements in his study are presented in Table 4.9, along with the conservative acceptable air concentration standards developed by a panel of toxicologists chaired by P. E. Morrow (Morrow et al. 1977). Morrow et al. used the data of Vaughn et al. (1975), ambient air concentrations, atmospheric standards for population exposures, reference man data for inhalation intakes, and occupational Threshold Limit Values to establish safe ranges of atmospheric concentrations.

During combustion, trace elements tend to variably concentrate in three fractions that ultimately effect their emission: Group I--those that are not volatilized, but melt and become bottom ash and slag or are emitted from the stack as fly ash; Group II--those that are volatilized but condense out in the fly ash particles as the flue gas cools; and Group III--those that are volatilized and remain almost completely in the gas phase (Klein et al. 1975; Ray and Parker 1977). The distribution of these trace elements is shown in Table 4.10.

Attempts at quantifying trace element emissions are troubled by errors in sampling and analysis methods. Cowherd et al. (1975) found an average of 50% more trace material in raw coal samples than in all the effluent streams combined. Comparison of the work of Ray and Parker (1977), Natusch et al. (1974), and Magee et al. (1973) reveals substantial inaccuracies and variations in the data (U.S. Office of Technology Assessment 1979). This inadequacy of the data has lead the Office of Technology Assessment (1979) to conclude that a reasonable estimate of mass emission rates of trace pollutants cannot be made at present.

4.3.2 Summary of Health Effects

Trace elements in ambient air are primarily associated with particulate matter (Friedlander 1973). Natusch et al. (1974) found that arsenic, antimony, cadmium, lead, selenium, and thorium were most concentrated on the smallest respirable particules due, in theory, to preferential adsorption. These particles, the most difficult to control using pollutant removal equipment, are potentially the most hazardous to human health. A complete discussion of respirable particulates appears in Section 4.1.

Speculation regarding the ability of fine particulates to impact human health by penetrating the lung's defenses and exposing it to concentrated toxic materials on the surface of the particles is based on the same type of controversial epidemiological studies that have implicated sulfate as being associated with premature deaths (U.S. Office of Technology Assessment 1979). Obviously, inhalation is the most significant route of exposure from these air pollutants and the lung the major target organ. As noted in Section 4.1, the 1977 Clean Air Act amendments require the U.S. Environmental Protection Agency (USEPA) to examine these potential health effects and to establish regulatory controls for fine particulates and associated trace elements if necessary.

Table 4.8. Distribution of Some Potentially Hazardous Trace Elements in Coal (ppm in coal)

	Region			
	Powder River	Western Interior	Eastern Interior	Appalachian
Antimony	0.67	3.5	1.3	1.2
Arsenic	3	16	14	18
Beryllium	0.7	2	1.8	2.0
Cadmium	2.1	20	2.3	0.2
Mercury	0.1	0.13	0.19	0.16
Lead	7.2		34	12
Selenium	0.73	5.7	2.5	5.1
Zinc	33		250	13

From Zubovic (1975), Table 3, p. 12A.

Table 4.9. Predicted Air Concentrations of Airborne Trace Elements in the Vicinity of a 1400-MWe Coal-Fired Powerplant Compared with Acceptable Air Concentrations

Element	Predicted Air Concentrations[a] ($\mu g/m^3$)	Acceptable Air Concentrations[b] ($\mu g/m^3$)
As (V) or total	1.2×10^{-4}	0.1
As (III)		1×10^{-5}
Be	2.9×10^{-4}	5×10^{-3}
Cd	1.2×10^{-4}	0.05
Cr	7.2×10^{-6}	0.05
Cr (VI insol)		1×10^{-6}
F	2×10^{-4}	0.01
Hg	1.2×10^{-3}	0.1
Hg-organic		0.01
Mo	2.4×10^{-4}	0.1
Ni	4.8×10^{-4}	0.01
Ni carbonyl		1×10^{-6}
Pb	2.4×10^{-4}	1.0
Sb	2.4×10^{-5}	0.1
Se	2.4×10^{-5}	0.1
Tl	3.0×10^{-6}	0.01
U	3.0×10^{-3}	0.01
V	2.4×10^{-4}	0.05

From Morrow et al. (1977).

[a]Values taken from Vaughan et al. (1975) except for F and U. The F value is based on an assumed 80 ppm of F in coal and a 50% volatile fraction, of which 33% is associated with particulate matter. No estimate was made of gaseous F levels. The U value was derived from Martin et al. (1971).

[b]Values based on sources listed in Morrow et al. (1977). Where human intake data were used, ambient air concentrations were calculated assuming 10 m^3 air/day breathed with 100% particulate deposition. Values shown represent consideration of urban and ambient air data. Where these data varied widely or were limited and the metal has a relatively short biological retention, additional weight was given to the 1/1000 occupational TLV.

Table 4.10. Distribution of Trace Elements in Coal Combustion Residues

Group I - Equally distributed in bottom ash and fly ash			
Barium	(Ba)	Manganese	(Mn)
Chromium	(Cr)	Rubidium	(Rb)
Cerium	(Ce)	Scandium	(Sc)
Cobalt	(Co)	Samarium	(Sm)
Europium	(Eu)	Strontium	(Sr)
Hafnium	(Hf)	Tantalum	(Ta)
Lanthanum	(La)	Thorium	(Th)
Group II - Preferentially concentrated in fly ash			
Arsenic	(As)	Lead	(Pb)
Beryllium	(Be)	Antimony	(Sb)
Cadmium	(Cd)	Selenium	(Se)
Copper	(Cu)	Uranium	(U)
Gallium	(Ga)	Vanadium	(V)
Molybdenum	(Mo)	Zinc	(Zn)
Nickel	(Ni)		
Group III - Discharged as vapors			
Chlorine	(Cl)	Mercury	(Hg)
Fluorine	(F)		

Adapted by U.S. Office of Technology Assessment (1979) from Ray and Parker (1977).

The trace element data in this compilation will deal with known health effects, actual and predicted exposures, recommended standards, etc. related to atmospheric emission. Some of the information has been arranged on logarithmic scales to allow graphic illustration of potential health effects. The data from various experiments using either single-celled organisms or biochemical fractions of organisms usually is depicted relative to a logarithmic scale of aqueous molar concentrations of the element concerned. Such data cannot be directly applied to quantitative human health estimation without detailed understanding of human target cells, human metabolic conversion and transport, interspecies extrapolation of biological knowledge, and environmental conversion and transport. On the other hand, such data can provide a basis for comparison among environmental toxicants and hence contribute to the ranking of toxic materials of concern in a site-specific environmental impact analysis.

Cellular and subcellular studies show that in many cases these elements are essential to the proper biochemical functioning of the human body. Sodium, potassium, and chloride ions are required for the proper function of nerve cells (Hodgkin et al. 1952). Almost one third of all enzymes require the presence of metal ions (Simkiss 1979). In contrast, other trace elements are toxic and have no known useful biochemical function. Examples include lead, mercury, and camdium (Fowler 1978). Finally, some elements such as zinc or manganese are nutritionally required at low doses while being toxic at higher doses.

Comparative studies may in some cases be very useful in identifying toxicants of greatest concern. Several experimental studies which compare several metal salts for various cellular effects are discussed here.

Paton and Allison (1972) in the course of their work determined the concentrations of various salts toxic to human leukocytes in culture. For the metals they tested there seems to be a dichotomous result. Antimony, selenium, mercury, cadmium, and arsenic are toxic at low doses while vanadium, cobalt, and beryllium are toxic only at comparatively high levels. Only tellurium is ambiguous, with one salt toxic at low levels and another one at high levels. These results are depicted in Figure 4.20.

Sirover and Loeb (1976) studied the effect of various metal salts on the fidelity of *in vitro* DNA synthesis. The metal salts were added to reaction mixtures in which DNA polymerase from avian myeloblastosis virus catalyzed the incorporation of nucleotides into polymers complementary to artificial polynucleotide templates. The only radioactively labeled nucleotide in the reaction mix was not complementary to any of the bases in the template. Radioactivity incorporated into polynucleotides is indicative of errors in DNA replication. Here, also, a few metals (chromium (II), zinc, cadmium, copper, and silver) elicited a response at much lower concentrations than did the bulk of the salts tested. The results of this study are presented in Figure 4.21.

Korman et al. (1978) have studied the inhibitory effects of metal salts on the activity of *Micrococcus luteus* DNA polymerase I *in vitro*. Various concentrations of the metal salts were added to reaction mixtures containing calf thymus DNA as a template and tritiated thymidine triphosphate or tritiated adenosine triphosphate. The time rate at which tritiated polynucleotides appear in the reaction mix is a measure of the enzymatic activity. The concentration of metal salt resulting in a 50% reduction compared to controls was noted. Those results are shown in Figure 4.22. The metals fell into three classes: zinc, lead, and mercury are inhibitory at low concentrations; cadmium, nickel, cobalt, copper, and calcium inhibit at somewhat higher concentrations; and barium did not cause 50% inhibition at any level tested.

Results from several other comparative studies are depicted in Figure 4.23. Deknudt and Deminatti (1978) determined the concentrations of zinc chloride, lead acetate, and cadmium chloride that prevented cell division in human lymphocytes cultured *in vitro*. Waters et al. (1975) determined the concentrations of various salts leading to 50% survival of rabbit alveolar macrophages. Castranova et al. (1980) observed the metal salt concentrations leading to maximum and half maximum reduction in oxygen consumption by rat alveolar macrophages. The minimum salt concentration preventing formation of yeast colonies on agar plates was determined by Egilsson et al. (1979).

The overall evaluation of the cellular effects of heavy metals is a complex issue. Physicochemical factors, such as oxidation state and covalent bonding of the metal ion, affect its biological efficacy. Conjoint exposure to nonmetal toxicants can affect cellular response to metals. Finally, many of the metals exhibiting deleterious effects are nutritionally required. In some cases the nutritionally required level is not much below levels at which toxic effects are evident. Only better understanding of physiological effects of all environmental toxicants will ameoliorate the situation.

4.3.2.1 Arsenic

Arsenic (As) has been singled out as one of the potentially hazardous trace elements emitted from coal combustion (Friberg et al. 1977; Schroeder 1971; Lim 1979; Van Hook 1979; and Fishbein 1976).

ORNL-DWG 81-6631

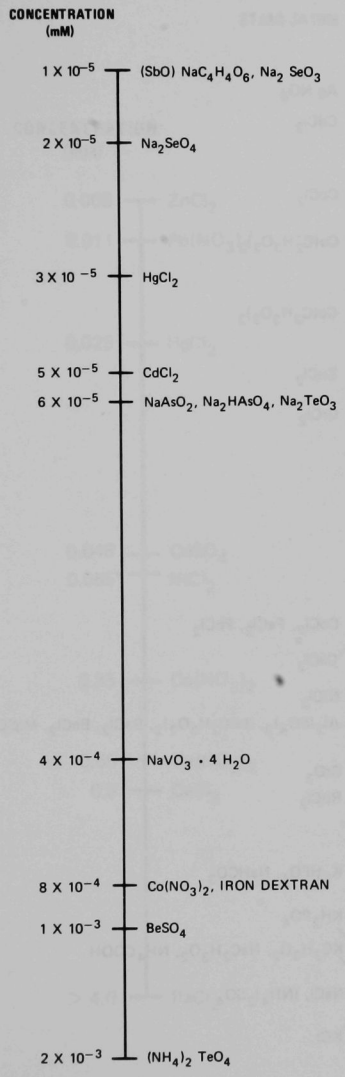


Fig. 4.20. Toxic Metal Salt Concentrations for Human Leukocyte Cultures

ORNL-DWG 81-6629

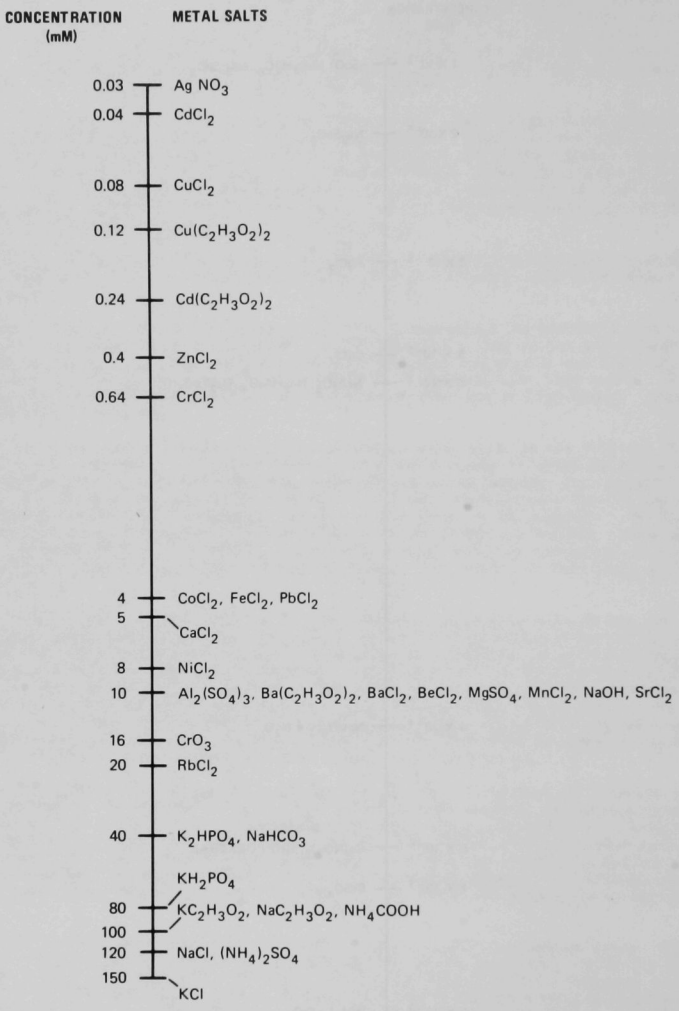


Fig. 4.21. Metal Salt Concentrations for Maximum Infidelity of DNA Synthesis

ORNL–DWG 81-6630

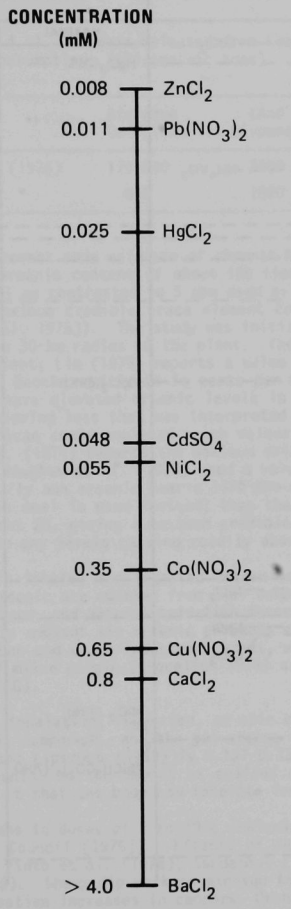


Fig. 4.22. Metal Salt Concentrations for 50% Inhibition of DNA Polymerase

ORNL-DWG 81-6628

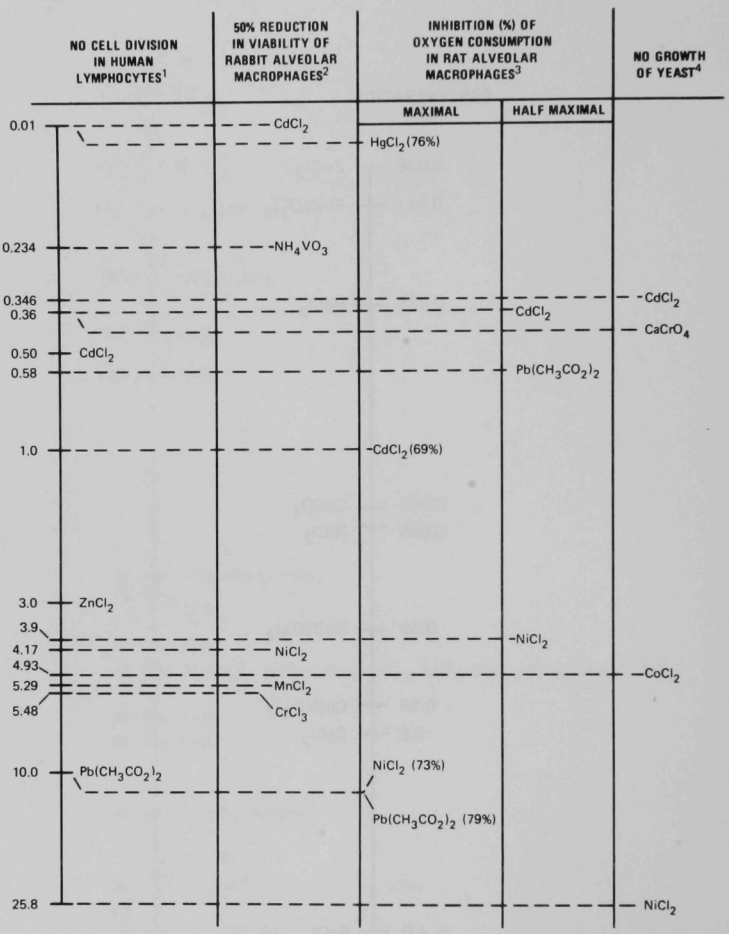


Fig. 4.23. Inhibitory Metal Salt Concentrations in Other Studies

Two estimates of the total amount of arsenic released during coal consumption are given in Table 4.11. Primary smelting (~50%), use of pesticides (~32%), and fossil-fuel combustion (~6%) comprise the three major sources of arsenic releases (Friberg et al. 1977; Suta 1980). One of the four major sources of arsenic in air is burning coal, since coal contains 0.08-16 μg/As/g coal (Vallee 1973). Mainly in particulate form as arsenic trioxide and metal arsenites (Friberg et al. 1977), arsenic concentrations in air are very low and often below detectable limits except in the vicinity of smelters. The average daily concentration for 133 locations in the U.S. was 0.02 $\mu g/m^3$, with a few higher yearly values of ~0.8 $\mu g/m^3$ (National Air Surveillance Network [NASN] 1964, 1966). Ambient air levels of arsenic appear to be dropping, reflecting a reduction in particulate emissions from commercial arsenic sources (Thompson 1976). There are no data on health effects below the 0.2 $\mu g/m^3$ level (Fishbein 1976).

Table 4.11. Arsenic Released from Coal Consumption, 1974 (metric tons)

Reference	Airborne Emissions	Land Disposal	Total
Holt and Moberly (1976)	170-340	3000	3350
Vesar (1976)	650	1800	2450

Bencko and Syman (1977a, 1977b) present some evidence of chronic health effects due to burning Czechoslovakian brown coals with arsenic content of about 100 times the average American coal content (900-1500 ppm on Czech coal as contrasted to 5 ppm used as the average for western coal in Vaughan's calculation of the maximum credible trace element concentration from a 1400-MWe coal-fired powerplant [Vaughan et al. 1975]). The study was initiated to determine the cause of mass extinctions of honey bees in a 30-km radius of the plant. They determined that high levels of arsenic were present near the plant; Lim (1979) reports a value of 69 $\mu g/m^3$, probably calculated by Lim from data presented by Bencko and Syman in parts per million. Children near the plant were examined and found to have elevated arsenic levels in their hair and urine. Some evidence was found suggestive of hearing loss that was interpreted to be due to arsenic exposure. Only a very small portion of American coals analyzed reach values up to 10% of the Czech coal values (Zubovic 1975). Ruch et al. (1974) reports the maximum value found in an analysis of 82 Illinois basin coals as 93 ppm. Vaughan et al. (1975) used a value of 5 ppm in predictions of maximum credible concentration of fly ash arsenic near a 1400-MWe coal-fired powerplant; if the value of 93 ppm from high Illinois coal is used instead, then the predicted output of arsenic should increase by a factor of about 20, giving a maximum credible value of 0.0024 $\mu g/m^3$, which is still very low in comparison to any levels causing readily observable human health effects.

Since the chemical form of arsenic inhaled and ingested determines its toxic effects, it is important to know what forms of arsenic are emitted from coal combustion. The pentavalent form is less toxic than the trivalent form, and natural oxidation favors the conversion of trivalent arsenic to pentavalent arsenic. In ambient air arsenic probably exists in the following forms: inorganic forms dominate in suburban and urban/industrial areas, with both tri- and pentavalent arsenic detected in air samples of mixed origin (Crecelius 1974) and primarily trivalent arsenic in smelter air samples (Andreae 1980).

Arsenic poisoning can result from inhalation, ingestion, or skin contact with the element, some forms of the inorganic and organic compounds, and the gas arsene (Lim 1979). The daily intake for humans calculated by Duggan and Lipscome (1969) is 0.137-0.33 mg/person; others suggest a range of 0.15-0.4 mg/person, depending on the amount of seafood eaten (Fishbein 1976; Smith 1972). Friberg et al. (1977) report that the body can tolerate from 14-20 mg/d.

Arsenic probably is fatal to humans in doses of 1 to 10 g (calculated from LD_{50} data given for animals by the National Research Council [1976]). Effects of lesser doses are reviewed in National Research Council (1976), Pinto et al. (1976), Sunderman (1976), Blackwood et al. (1979), Beliles (1975), and Fishbein (1976). Sheep dip workers exposed to 254-696 $\mu g/m^3$ arsenic showed small, but epidemiologically suggestive increases in cancers. Orchard sprayers exposed to 140 $\mu g/m^3$ lead arsenate and industrial workers exposed to 100 $\mu g/m^3$ less than 25 years showed no increases in cancers (National Research Council 1976; Pinto et al. 1976). While these data indicate an epidemiologically significant increase in cancers in workers exposed to approximately 250 $\mu g/m^3$ or greater arsenic, the total potential carcinogen exposure of the workers is not considered in the correlation. These and other studies point to the carcinogenicity of arsenic (U.S. Dept. of Labor 1975), but suggest that other cocarcinogenic factors must be examined before any conclusions are reached (NIOSH 1973a). To date there has been no convincing evidence of the carcinogenicity of arsenic in experimental animals (U.S. Dept. of Labor 1975; Sunderman 1976).

Epidemiological studies have shown a small correlation between arsenic and increased cancer risk when arsenic is present in air at concentrations roughly 2.5 million times higher than those

predicted near a coal-fired plant. Similar studies with arsenic concentrations one million times higher than those predicted near a coal-fired powerplant do not show any increased cancer risk. Arsenic has not been demonstrated to be carcinogenic in any laboratory animal. All other health effects require even higher exposures than the suggested carcinogenic levels. A maximum credible daily human uptake rate from coal would be 0.0024 μg/day.

One of the more conservative approaches to assessing the potential impact of arsenic from coal combustion is that done by a panel of 15 scientists chaired by P.E. Morrow (Morrow et al. 1977). They suggest an "acceptable air concentration" value of 1×10^{-5} μg/m^3 for arsenic (III), which would mean that the predicted maximum credible air concentration from fly ash (Vaughan et al. 1975) of 1.2×10^{-4} μg/m^3 would be roughly 10 times higher than an acceptable amount. Morrow's panel had no data available for arsenic (III) potential carcinogenicity so they used a proposed standard for chromium (IV) that used 10^{-2}% of the ambient level. Many of the above data are summarized in Figure 4.24.

Fowler (1978) has reviewed the subcellular effects seen when arsenic is administered to animals. Chromosomal aberrations occur more frequently in lymphocytes of workers exposed to high levels of arsenical compounds. Such compounds also inhibit mitochondrial respiration, uncouple oxidative phosphorylation, and increase levels of mitochondrial marker enzymes such as monoamine oxidase and cytochrome oxidase. Prolonged oral exposure to arsenic has very little, if any, effect on microsomal enzyme levels.

Paton and Allison (1972) studied induction of chromosomal aberrations in cultures of human leucocytes. They found that both sodium arsenate and sodium arsenite are toxic to such cultures at a concentration of 6.0×10^{-8} M. On the other hand, only the trivalent (arsenite) form, at concentrations between 2.9×10^{-9} and 1.8×10^{-8} M, induced chromatid breaks. Nishioka (1975) tested various arsenical salts for different toxicities in DNA-repair-proficient and -deficient strains of the bacterium Bacillus subtilis. Again, sodium arsenite showed the biggest effect. Tkeshelashvili et al. (1980) tested two pentavalent arsenic salts and found that neither caused elevated rates of misincorporation in in vitro DNA synthesis experiments. Leonard and Lauwerys (1980) have reviewed the literature on arsenic mutagenicity. These effect levels are summarized in Figure 4.25.

4.3.2.2 Beryllium

Beryllium (Be) is one of several trace elements emitted chiefly from coal combustion (Friberg et al. 1977; Schroeder 1971). With increasing use of coal for space heating and production of electricity, greater concern has been expressed regarding the beryllium-containing particles being emitted in the stack effluents (Tepper 1972a,b). The tremendous variability in beryllium content in U.S. coals makes it difficult to estimate future releases from coal-burning utilities. However, beryllium has been found in flue dusts at an average concentration of 20 ppm. Although the chemical form of the beryllium in the effluent has not been characterized, it is likely to be BeO (Tepper 1972a,b).

Phillips (1973) states that 84% of the beryllium in coal is released in the stack gas; however, later analysis by Gladney and Owens (1976) indicates that 95% of the beryllium in coal is retained in the fly ash removed from the stack gas. Gladney and Owens (1976) could not measure beryllium in the ambient aerosols near a coal-fired powerplant, but found about 1.1 μg/m^3 in the stack gas, with selective enrichment in the smaller particles.

Beryllium, a highly toxic metal, is retained by the lungs following inhalation and is slowly mobilized. It is not readily absorbed by any route of exposure and only about 1% of inhaled beryllium is absorbed. Long-term industrial exposure to beryllium compounds leads to berylliosis, a chronic pulmonary disorder with a 30% mortality rate (Tepper 1972a,b). For example, industrial exposure of 0.31-1310 μg/m^3 caused chronic lung disease, including radiographic lung abnormalities in 31 of 214 workers; 11 of the 214 had hypoxia (Kanarek et al. 1973). Concern over potential harm to workers handling beryllium has led to the creation of the U.S. Beryllium Case Registry, which listed 577 chronic beryllium disease cases by 1972, most occurring before standards reduced exposure (Drury et al. 1978).

Epidemiological analysis of beryllium workers has failed to demonstrate any correlation with increased cancer risks (International Agency for Research on Cancer 1972). While cancers have been produced in rats inhaling some beryllium compounds (Drury et al. 1978), no counterparts to these experimentally induced cancers have been found in humans (U.S. Dept. of Health, Education and Welfare 1972). Ingested beryllium also damages the skeleton and lungs (Lim 1979). Beryllium toxicity has been reviewed in greater detail by Bowen (1966), Drury et al. (1978), Aldridge (1966), Tabershaw (1972), Higgins (1968), Preuss (1975), and Tepper (1972a,b).

Except in unusual situations, the public is not at risk from beryllium. Beryllium is undetectable in the atmosphere over most U.S. cities (USEPA 1973a). Bowen (1966) estimates beryllium concentrations to be <0.0001 μg/m^3 in the atmosphere. Concentrations as high as 0.01 μg/m^3 have been measured in neighborhoods near beryllium-using industries (Utidjian 1973; Cholak et al. 1962).

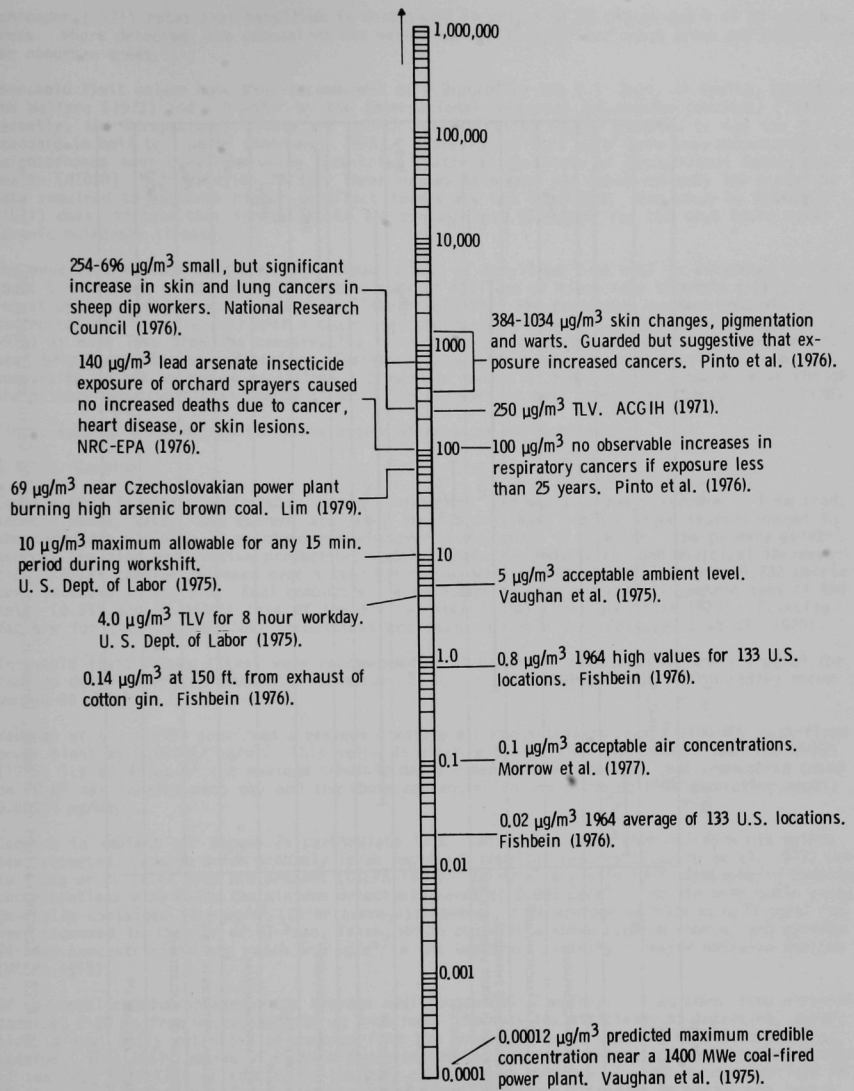


Fig. 4.24. Levels and Effects of Arsenic (As)

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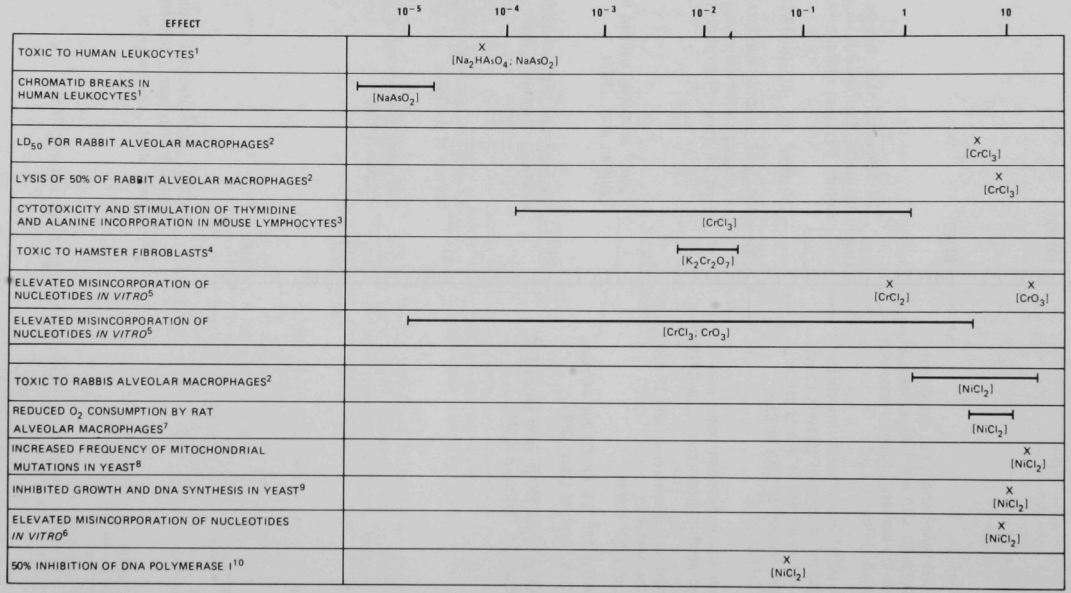


Fig. 4.25. Concentrations of Arsenic, Chromium, and Nickel Causing Cellular Effects (mM)

Schroeder (1971) notes that beryllium is detectable in only 9 of 58 cities and 4 of 29 nonurban areas. Where detected, the concentrations were 0.001-0.002 μg/m^3 for urban areas and 0.00013 μg/m^3 for nonurban areas.

Threshold limit values have been recommended at 0.2 μg/m^3 by the U.S. Dept. of Health, Education and Welfare (1972) and 2.0 μg/m^3 by the International Technical Information Institute (1975). Recently, the Occupational Safety and Health Administration (OSHA) proposed to cut the TLV standard in half to 1 μg/m^3 (Anonymous 1975). Standards of 0.01 μg/m^3 have been recommended for neighborhoods near beryllium-using industries (National Institute of Occupational Safety and Health [NIOSH] 1972; Utidjian 1973). These values have been set conservatively low since the data required to estimate higher no-effect levels are not available. One study by Schroeder (1971) does indicate that inhalation of air containing 0.002 μg/m^3 for 250 days could cause chronic pulmonary illness.

The predicted maximum credible daily human uptake of beryllium from coal is estimated to be about 6×10^{-5} μg, on the order of several tens of millions of times less than the average daily ingestion rate of 100 mg estimated by Fishbein (1976). The predicted maximum credible air concentration (from fly ash) near a coal-fired powerplant of about 3×10^{-6} μg/m^3 (Vaughn et al. 1975) is much less than the conservative recommended standard of 0.01 μg/m^3 for neighborhoods near beryllium-emitting industries. Therefore, atmospheric emissions of beryllium from coal combustion do not appear to constitute a present or potential human health problem, even though the primary nonoccupational source of beryllium exposure is coal combustion (Drury et al. 1978).

Figure 4.26 is a summary of the above health effects and exposure data.

4.3.2.3 Cadmium

Cadmium (Cd) is widely distributed in the environment, and man's primary exposure is from food, tobacco smoke, water, and ambient air. Food and tobacco smoke are the major sources except in the immediate vicinity of point-source atmospheric emissions of cadmium. The primary point-sources are zinc and cadmium production, cadmium consuming industries, and municipal incineration. Total cadmium released over a ten-year period was 140,519 metric tons, with 9,732 metric tons released to the air. Coal combustion, a secondary source, produced 740 metric tons of the total (0.5%) and 69 metric tons of the air releases (0.7%) (Yost and Miles 1979). Emission factors for cadmium from fuel combustions are shown in Table 4.12 (Blackwood et al. 1979).

Threshold limit values (TLVs) were recommended at 50 μg/m^3 for cadmium fumes and 150 μg/m^3 for cadmium dusts (American Council of Governmental Hygienists [ACGIH] 1976). NIOSH (1976) recommended 40 μg/m^3.

Vaughan et al. (1975) predicted a maximum credible air concentration near a 1400-MWe coal-fired power plant at 0.000012 μg/m^3. This value is roughly 3.3 million times lower than the NIOSH (1976) TLV of 40 μg/m^3. A maximum credible daily human uptake rate from coal combustion based on 20 m^3 air inhaled each day and the above concentration and assuming 100% absorption equals 0.00024 μg/day.

Cadmium in ambient air occurs in particulate form. While its exact chemical form has seldom been reported, cadmium oxide probably is an important chemical species (Friberg et al. 1977) and sulfides or sulfates also are present (USEPA 1975). In rural areas in 1969, atmospheric cadmium concentrations were below the minimum detectable level of 0.003 μg/m^3. The air over urban areas generally contained >0.1 μg/m^3 (24-hr average); however, a 24-average as high as 0.73 μg/m^3 has been measured in the air of El Paso, Texas, which contains a known cadmium source, and average 24-hour concentrations may reach 5-6 μg/m^3 in the immediate vicinity of major emission sources (USEPA 1975).

Of the total cadmium intake by the average adult human (50-75 μg/day), <1 μg comes from airborne cadmium, 2-20 μg from water, and ~50 μg from food. Because the efficiency of intestinal absorption is low, daily retention of cadmium from the above levels of exposure equals only 1-2 μg cadmium. This daily uptake of cadmium approximates the net daily increase in body burden because of the long half-life of cadmium in the body. The strong tendency of cadmium to accumulate in the body is sufficient cause for concern over possible future increases in background levels (Hammons et al. 1978).

Respiratory absorption is usually an important pathway into the human body. Few data exist on the deposition, retention, and elimination of cadmium aerosol. Assuming that inhaled cadmium behaves in the respiratory system in much the same way as do other particulates, the amount of cadmium inhaled depends on the volume of air (average inhalation = 20 m^3/day) and the ambient concentration of cadmium (USEPA 1975). The fraction of the inhaled cadmium deposited in the lung depends on the particle size (see Sec. 4.1). Available data indicate that ~40% of the cadmium is in particles >2 μ in diameter; therefore, a large portion of the particles would be in the respirable range (USEPA 1975). Absorption occurs primarily in the lungs, but also occurs in the gastrointestinal tract after mucociliary clearance. Data on animal experiments suggest

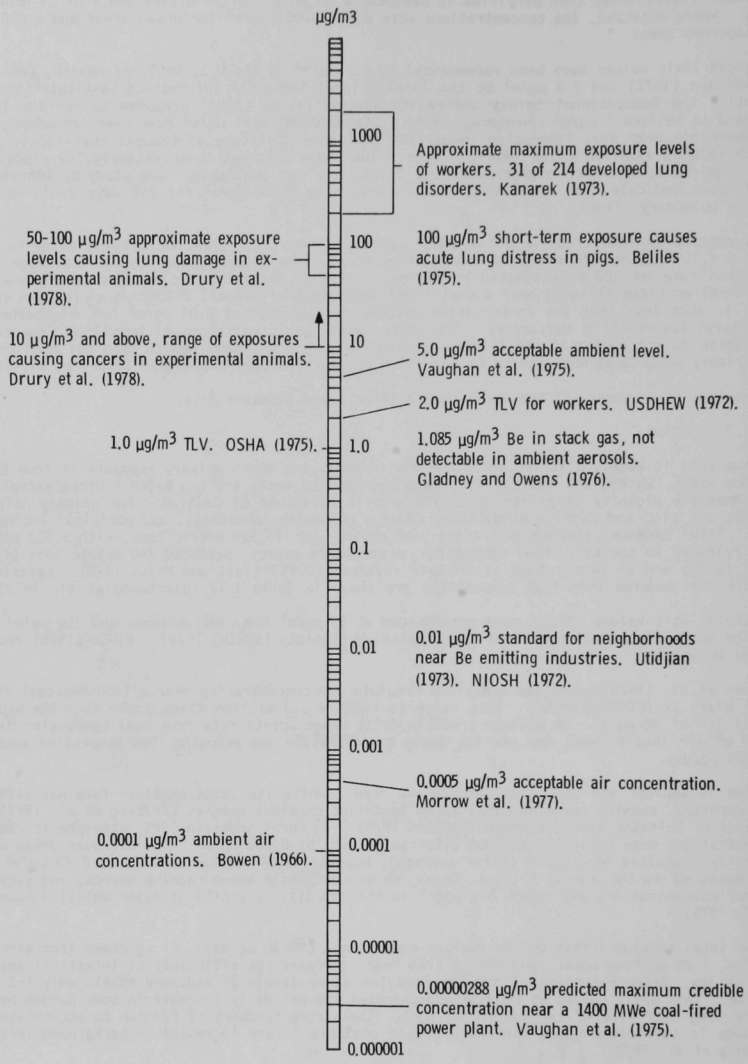


Fig. 4.26. Levels and Effects of Beryllium (Be)

Table 4.12. Emission Factors for Cadmium from Fuel Combustion

Source	Cadmium Content (ppm)	Emission Factor
Heating oil (residual assumed)	0.4 to 0.5	4 to 0.05[a]
Diesel oil	0.07 to 0.1	0.09 to 0.07[a]
No. 6 residual fuel oil		
Virgin Islands	5	5[a]
Virgin Islands[b]	<0.4	-[c]
Curacao	4	4[a]
Trinidad	3	3[a]
Venezuela	<0.4	-
Coal		
Kansas powerplant	-	0.1[d]
Michigan powerplant	-	0.5[d]

From Blackwood et al. (1979b).

[a] g/m^3 of oil consumed.

[b] Different oil fields.

[c] A dash indicates that cadmium content is below limits of detection.

[d] g/metric ton of coal burned.

that ~10 to 50% of inhaled cadmium is absorbed, depending on the concentration, particle size, and solubility of the particle (Friberg et al. 1977). A retention rate of 11% is not unreasonable for urban levels of exposure.

Acute effects of inhalation of cadmium oxide (CdO) or cadmium chloride ($CdCl_2$) aerosols have been observed in animal studies in three stages: (1) acute pulmonary edema, within 24 hrs of exposure; (2) proliferative insterstitial pneumonitis, observed 3-10 days after exposure, and (3) permanent lung damage in the form of perivascular and peribronchial fibrosis. The first two stages have been confirmed clinically or through autopsy for humans (Friberg et al. 1971).

Chronic inhalation exposure is characterized by proteinuria from kidney damage and emphysema from lung damage (Beliles 1975). Cadmium oxide fumes are more hazardous than cadmium-containing dusts. Eight-hour doses of cadmium oxide fumes at 1000 $\mu g/m^3$ are immediately hazardous, and eight-hour doses at 5000 $\mu g/m^3$ probably are fatal. Cadmium dust exposures at levels of 3000 to 15000 $\mu g/m^3$ over 20 years may cause some lung damage (emphysema) (Friberg et al. 1974). Actual exposures of 30-690 $\mu g/m^3$ to cadmium stearate and lead dusts produced no observable effects (Suzuki et al. 1965). Lauwerys et al. (1974) estimated that an exposure of 21 $\mu g/m^3$ for 20 years would cause renal damage. Fleisher et al. (1974) estimated that 1-2 $\mu g/m^3$ would induce renal cortex damage after 50 years of exposure. Using data from workers exposed to cadmium and from animals manifesting functional and morphological changes in the renal cortex following exposure to cadmium, Friberg et al. (1971, 1974, 1975) have concluded that a cadmium concentration of about 200 ppm (wet weight) in the renal cortex is a "critical concentration," causing the first sign of tubular dysfunction (tubular proteinuria) to appear in sensitive persons. Estimates of the long-term exposure necessary to achieve a concentration of 200 ppm of cadmium in the renal cortex are given in Table 4.13.

The estimated minimum cadmium levels via inhalation that are necessary to reach a potentially damaging level (200 ppm, wet weight, of cadmium in the renal cortex) are shown in Table 4.13 to be 0.08 $\mu g/m^3$ at a 40% retention rate over 50 years in ambient air.

Animal experiments also link anemia, hypertension, testicular necrosis, and carcinogenesis with cadmium exposure (USEPA 1975). In the cases of anemia, cardiovascular disease and hypertension, and carcinogenesis, animal studies have indicated an association with cadmium exposure, and epidemiological studies have been attempted in order to confirm these associations in humans.

Epidemiological studies correlating dustfall data or cadmium concentrations in air with hypertension and arteriosclerotic heart disease offer some equivocal data but no firm evidence (USEPA 1975; Friberg et al. 1971, 1974, 1975). Studies by Carroll (1966), Hickey et al. (1967), and Hunt et al. (1971) are reviewed in detail in Friberg et al. (1971, 1974, 1975) and their ambiguities are detailed. Hammer et al. (1972) studied groups of workers with varying exposure to

Table 4.13. Estimated Minimum Cadmium Levels via Inhalation or Ingestion Necessary for Reaching 200 ppm (wet weight) of Cadmium in Renal Cortex (total body burden: 120 mg cadmium) ($\mu g\ Cd/m^3$)

Exposure (years)	Total Daily Ingestion			Ambient Air[a]			Industrial Air[a]		
	Retention Rate (%)			Retention Rate (%)			Retention Rate (%)		
	2.4	5	10	10	25	40	10	25	40
10	1324	662	331	16.2	6.5	4.1	52.2	21.0	13.1
25	530	265	132	6.5	2.6	1.6	21.0	8.4	5.2
50	265	132	66	3.2	1.3	0.8	10.4	4.2	2.6

From Friberg et al. (1971, 1974, 1975).

[a]A lung ventilation of 20 m^3 per day has been used for evaluation of ambient air exposure. A lung ventilation of 10 m^3 per 8 hours for 225 days per year has been used for evaluation of industrial air exposure. No corrections have been made for cumulative effects of different types of exposure, including tobacco smoking. A linear approximation of the accumulation of cadmium has been used.

cadmium but could not find a consistent relationship between cadmium and blood pressure. At least three studies of workers with long-term exposure to cadmium showed excess cancer deaths (Potts 1965; Kipling and Waterhouse 1967; Lemen et al. 1976; see also Friberg et al. 1950). The International Agency for Research on Cancer (1976) evaluated the association between occupational cadmium exposures and cancer, stating:

> "Available studies indicate that occupational exposure to cadmium in some form (possibly the oxide) increases the risk of prostate cancer in man. In addition, one of these studies suggests an increased risk of respiratory tract cancer."

Even the epidemiological studies on Itai-Itai disease in Japan, thought to be caused by long-term ingestion of rice contaminated by cadmium from river water used for irrigating rice fields, are not conclusive as to cause and effect between cadmium-containing effluents and human health (Fulkerson et al. 1973). This very painful degenerative bone disease affected at least 200 people, of which nearly half died by 1965. A short review of the clinical and epidemiological data for Itai-Itai is given in USEPA (1975) and a detailed review appears in Friberg et al. (1971, 1974, 1975).

Exposure levels and some health effects of cadmium are summarized in Figure 4.27.

Cadmium-induced mutagenesis has not been demonstrated (Hammons et al. 1978), but cellular and subcellular studies indicate cells with chromosomal aberrations and other cell-specific effects of cadmium.

When rodents are exposed to ionic cadmium, several changes in cellular biochemistry become evident (Singhal and Merali 1979). Administration of cadmium chloride to rats results in higher blood glucose levels and elevated hepatic gluconeogenic enzyme levels. Cadmium ion in rats will raise hepatic and cardiac levels of both cyclic adenosine monophosphate and adenylate cyclase activity. In addition, cadmium ion reduces glucose tolerance, serum insulin levels, and levels of cardiac mitochondrial oxidative phosphorylation in rats. Exposure of mammals or mammalian cultured cells to cadmium, zinc, silver, or mercury ions results in de novo synthesis of metallothionien. This protein will bind the above ionic species as well as copper and tin (Probst 1979).

Various effects have been noted in whole cells derived from humans. Deknudt and Leonard (1975) found a significant increase over background in the frequency of chromosome aberrations in leukocytes of men occupationally exposed to cadmium fumes. Shiraishi et al. (1972) found that human leucocytes incubated eight hours in the presence of 4.3×10^{-7} molar (M) cadmium sulfide (CdS) exhibited higher frequency of aneuploid cells and of cells with chromosomal aberrations. Deknudt and Deminatti (1978) found that 5×10^{-4} M $CdCl_2$ in the growth medium completely blocked cell division of human lymphocytes. A lower concentration, 5×10^{-5} M, inhibited cell division

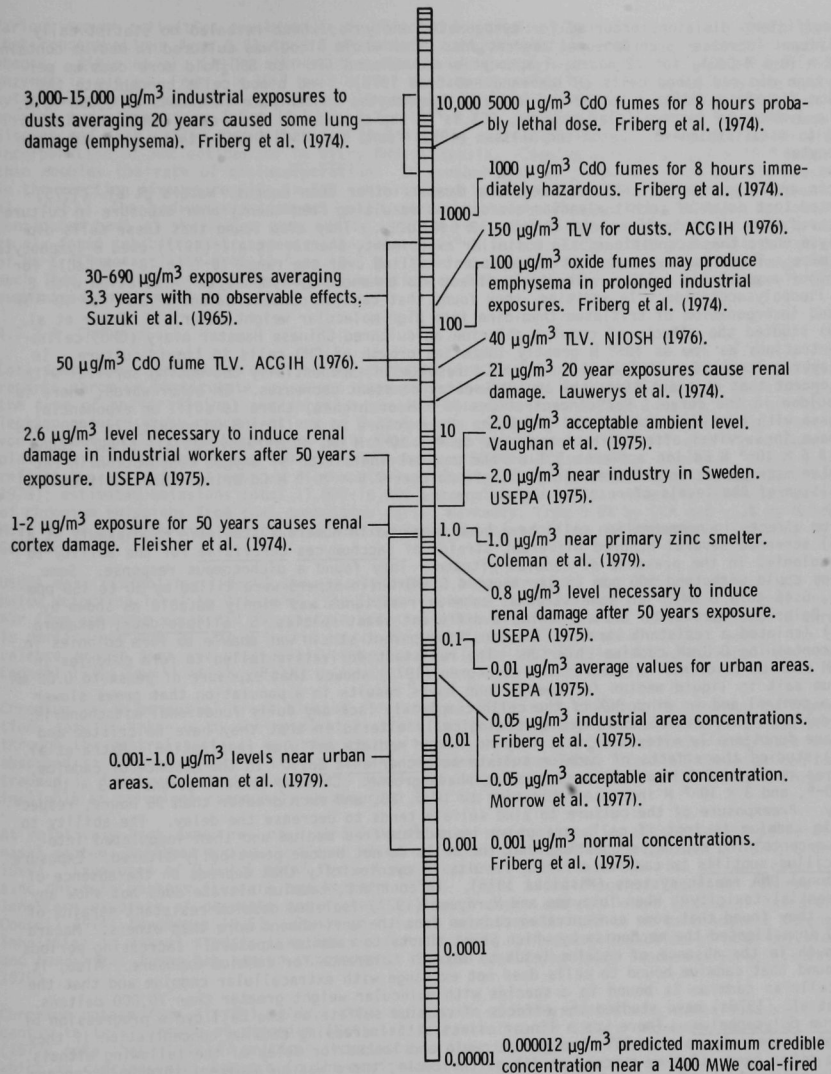


Fig. 4.27. Levels and Some Health Effects of Cadmium (Cd)

but sufficient divisions occurred for cytogenetic analysis, which revealed no statistically significant increase in chromosomal aberrations. When whole blood was cultured in medium containing 2×10^{-7} M $CdCl_2$ for 72 hours, lymphocytes accumulated 600- to 800-fold more cadmium per cell than did red blood cells (Hildebrand and Cram 1979). Red blood cells concentrate the cadmium five-fold relative to the medium while lymphocytes concentrate in excess of 3,000-fold. Of the lymphocyte intracellular cadmium, 96% is in the cytoplasm. This cytoplasmic cadmium is bound to metallothionein. Paton and Allison (1972) found 5×10^{-8} M $CdCl_2$ to be toxic to human leukocytes.

Effects are also seen in cells derived from mammals other than humans. Waters et al. (1975) reported that death of rabbit alveolar macrophages resulting from twenty-hour exposure in culture increased with concentration from 10^{-5} to 10^{-3} M $CdCl_2$. They also found that these cells did not lyse under these conditions. In a similar experiment, Shenker et al. (1977) used B lymphocytes from mice. They found a concentration-dependent killing over the range 10^{-7} to 10^{-3} M $CdCl_2$ for a 72-hour exposure in culture. The toxic effect was enhanced by lymphocyte stimulation with E. Coli lipopolysaccharide. In addition, they found that cadmium concentrations of 10^{-4} or 10^{-3} M blocked incorporation of tritiated thymidine into high molecular weight material. Enger et al. (1979) studied the effects of cadmium chloride on cultured Chinese Hamster ovary (CHO) cells. Concentrations as low as 10^{-6} M greatly inhibited growth of CHO cells in liquid culture. In monolayer culture, 2×10^{-7} or 4×10^{-7} M Cd results in exponentially decreasing survival with time except that at short times the apparent decay constant decreases. In other words, there is a shoulder in the curve. For concentrations 10^{-6} M or higher, there is still an exponential decrease with time, but there is no shoulder. Preexposure to 2×10^{-7} M Cd results in 1000-fold increase in survival after 24-hour exposure to 4×10^{-6} M Cd. Castranova et al. (1980) found that 3.6×10^{-4} M Cd ion achieves 50% of the maximal inhibition of oxygen consumption in rat alveolar macrophages in culture. They also found that 7.6×10^{-5} M Cd brings about half maximal inhibition of the levels of reactive oxygen species.

Cadmium effects in nonmammalian cells have been examined in numerous studies. Middlekauff et al. (1956) screened several hundred different strains of Sacchomyces cerevisiae for the ability to form colonies in the presence of cadmium nitrate. They found a dichotomous response. Some strains could withstand 500 ppm (i.e., about 4.5 mM) while others were killed by 50 to 150 ppm (i.e., 0.45 to 1.5 mM). In some strains, cadmium resistance was highly mutable as shown by Luria Delbruck fluctuation tests. Using a different yeast species, S. ellipsoideus, Nakamura (1963) isolated a resistant strain. The sensitive parent strain was unable to form colonies in agar containing 0.8-mM cadmium chloride. The resistant derivative failed to form colonies in 1.6-mM cadmium chloride. Lindegren and Lindegren (1973) showed that exposure of yeast to 0.09 mM cadmium salt in liquid medium for twenty-four hours results in a population that grows slower than a control and in which 30% of the cells completely lack any fully functional mitochondria. Such dysfunctional mitochondria are morphologically altered in that they have no cristae and they are functionally altered in that they no longer mediate cellular respiration. Mitra et al. (1975) studied the effects of cadmium sulfate on Escherichia coli. In the absence of cadmium, cultures required 5.5 hours to reach mid-log phase growth. Cadmium concentrations of 3×10^{-7}, 3×10^{-6}, and 3×10^{-5} M increased this time to 17.5, 23, and much greater than 96 hours, respectively. Preexposure of the culture to zinc sulfate tends to decrease the delay. The ability to grow in cadmium is lost if cells are grown in cadmium-free medium and then inoculated into cadmium-containing medium, suggesting that the cells do not become genetically altered. Exposure of Bacillus subtilis to cadmium chloride results in cytotoxicity that depends on the absence of functional DNA repair systems (Nishioka 1975). In contrast, cadmium nitrate does not show any differential toxicity. When Tokoyama and Murayama (1977) isolated cadmium-resistant strains of yeast, they found that some concentrated cadmium from the environment more than others. Macara (1978) investigated the mechanism by which yeast adapts to cadmium exposure. Increasing periods of growth in the absence of cadmium leads to loss of tolerance for cadmium exposure. Also, it was found that cadmium bound to cells does not exchange with extracellular cadmium and that the intracellular cadmium is bound to a species with molecular weight greater than 70,000 daltons. Chin et al. (1978a) have studied the effects of cadmium sulfate on the cell cycle progression of Physarum polycephalum. There was a linear effect with increasing cadmium concentration if they exposed plasmodial explants 75% through the cycle and looked for delay of the following mitosis. On the other hand, if they exposed at 45% of the cycle, there was an apparent threshold concentration below which there was little if any delay observed. A subthreshold exposure at 45% of one cycle provides protection against the expected effects of exposure at 75% of that cycle or at 75% of the subsequent cycle. In a companion paper (Chin et al. 1978b), they studied potential synergism between cadmium and other metals in this system. An early, subthreshold dose of cadmium worsened the effects of later exposures to cobalt, copper, lead, and zinc ions while it protected against the effects of mercury and nickel ions and had no effect on iron and magnesium ion exposures. Early, subthreshold exposure to cobalt, copper, lead, and zinc led to an increased effect of a later suprathreshold dose of cadmium. Similarly, mercury and nickel provided protection while Fe and Mn had no effect. Egilsson et al. (1979) tested cadmium chloride for its effect on yeast mitochondria and found that exposure to 0.818 mM results in three- to five-fold more respiration-deficient cells.

Various enzyme activities are known to be affected by cadmium ions. Waters et al. (1975) showed that concentrations from 0.01 to 1.0 mM inhibit acid phosphatase from rabbit alveolar macrophages. Aitio et al. (1978) studied the effects of several heavy metals on drug-metabolizing enzymes in vitro. From 2×10^{-6} to 5×10^{-4} M, cadmium iodide inhibited benzo(a)pyrene hydroxylation and ethoxycoumarin ethylation. From 4.3×10^{-5} to 4.3×10^{-4} M, cadmium iodide inhibited epoxide hydratase; from 1.7×10^{-5} to 4.3×10^{-4} M, it inhibited glutathione 5-tranferase. Sirover and Loeb (1976) found that 4×10^{-5} M $CdCl_2$ caused 35% increase over controls in misincorporation of nucleotides in in vitro DNA synthesis. Cadmium acetate at 2.4×10^{-4} M more than doubles the rate of misincorporation. In another system, 4.8×10^{-5} M Cd sulfate present in the reaction mixture results in 50% inhibition of the activity of DNA polymerase I (Korman et al. 1978). The sodium-potassium-activated adenosine triphosphatase (Na-K ATPase) has been considered part of the sodium pump and is inhibited by cadmium chloride (Lai et al. 1980). Lai et al. found inhibition at concentrations above 10^{-6} M in the reaction mix and complete inhibition of Na-K ATPase activity from 10^{-5} to 10^{-3} M. Cadmium chloride in this concentration range had a much less inhibitory effect on magnesium-activated ATPase activity. These effect levels are summarized in Figure 4.28.

4.3.2.4 Chromium

Coal combustion contributes much of the chromium (Cr) in urban air (Sullivan 1969a). Sullivan reports chromium particulate emissions from coal-fired plants from 2.3 to 31 ppm (depending on the type of boiler firing) and chromium gaseous emissions from 0.22-2.2 mg/m^3. Fly ash collection reduced these concentrations to 0.19-6.6 ppm and 0.018-0.5 mg/m^3, respectively. Lee and von Lehmden (1973) report chromium particulate emission concentrations for coal-fired power-plants ranging between 1 and 100 ppm, reflecting differences in coals. Tables 4.14 and 4.15 reflect two estimates of sources of chromium-containing emissions (GCA Corporation 1973; Goldberg 1973); estimated emissions total 11,000-16,000 metric tons per year. However, estimated percentages of chromium emissions from coal combustion varied markedly, from 8.6% by GCA and ~53% by Goldberg. Most of this difference can be attributed to the omission of the ferrochromium industry by Goldberg.

USEPA data (1973b) for 1968-69 show yearly average concentrations of chromium in urban air from below detection level to 0.1 µg/m^3 (the latter exceeded in only 59 of the 186 cities monitored). Air from nonurban areas did not contain detectable amounts of chromium. Most atmospheric chromium is particulate and is likely to lie in the trivalent state (Towill et al. 1977), one of the two valence states that are biologically important (III and VI - Beliles 1975; National Rsearch Council 1974; Towill et al. 1977).

Chromium is an essential trace element for humans. While most chromium is taken up by ingestion, some is taken up by the respiratory tract and through damaged skin. Absorption can occur through the gastrointestinal and respiratory tracts; however, natural chromium complexes are absorbed to a greater extent than trivalent chromium (Mertz 1967). Inhaled chromium can be trapped in the bronchi and subsequently swallowed (ingested), deposited in the alveoli (remains insoluble as trivalent compound), or absorbed into the bloodstream.

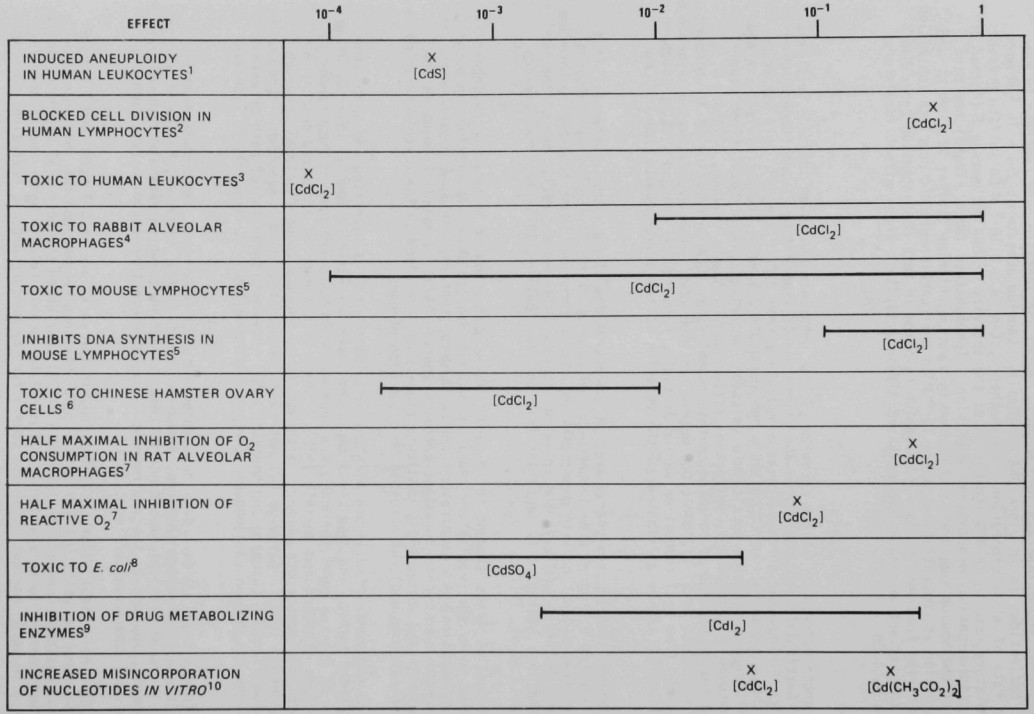
At relatively high exposures of 0.12-5.6 mg/m^3, some symptoms such as ulcerated and perforated nasal septum, inflamed mucosa, and nose bleed may occur (National Research Council 1974; Browning 1969). Exposures of 0.5-1.5 mg/m^3 for 6-9 years apparently increase the risk of lung cancer in zinc chromate workers (Langard and Norseth 1975). There is evidence of increased cancers in long-term exposure of chromite ore workers, although doses are not known (National Research Council 1974; Sunderman 1976). Chromium has not been demonstrated to be carcinogenic through inhalation in animal studies (Langard and Norseth 1975). However, injection of both trivalent and hexavalent forms has proven carcinogenic (Heuper and Payne 1962; Sunderman 1976; Fishbein 1976).

Chromium uptake from air (>1 µg/day) does not, under ordinary conditions, contribute significantly to total intake of chromium (National Research Council 1974). However, Morrow et al. (1977) note that it is of critical importance to know the extent to which hexavalent chromium, which is carcinogenic, appears in the environment. They believe that chromium (VI) probably does not occur in coal combustion effluents.

The ACGIH (1971) threshold limit value (TLV) for CrO_3 in the workplace is 0.1 mg/m^3 and 0.5 mg/m^3 for other soluable chromium salts. A TLV of 0.1 mg/m^3 has been recommended for some insoluble chromates (National Research Council 1974). NIOSH (1975) considered the potential carcinogenicity and recommended considerably lower standards at 1 µg/m^3 for poorly soluble mono- and dichromates and 25 µg/m^3 with a 50 µg/m^3 maximum 15-minute sample for the noncarcinogenic hexavalent chromates.

Many of the above health effects and exposure levels are summarized in Figure 4.29.

Chromium exposure in the workplace definitely is associated with increased incidence of lung cancer (Sunderman 1978). Cancer may occur at other sites as a result of exposure to chromium compounds and there is suggestive evidence that hexavalent compounds are the causative agents.



1. Shiraishi et al. (1972)
2. Deknudt and Deminatti (1978)
3. Paton and Allison (1972)
4. Waters et al. (1975)
5. Shenker et al. (1977)
6. Enger et al. (1979)
7. Castranova et al. (1980)
8. Mitra et al. (1975)
9. Aitio et al. (1978)
10. Sirover and Loeb (1976)

Fig. 4.28. Concentrations of Cadmium Causing Cellular Effects (mM)

Table 4.14. Sources and Estimates of Chromium-Containing Atmospheric Emissions in 1970

Source	Uncontrolled Emission Factor (kg/10^3 kg)	Production Level (metric ton/yr)	Chromium in Emissions (%)	Emissions of Chromium before Controls (metric ton/yr)	Estimated Level of Emission Control (%)	Emissions of Chromium after Controls (metric ton/yr)	Percent of Total U.S. Chromium Emissions
Mining							
None in United States				0			
Refining							
Ferrochromium							
Electric furnace	(100-415) 250[a]	341,000	22	18,700	40	11,200	68.2
Material handling	5	341,000	65	1,100	32	750	4.6
Electrolytic chromium	0.024	8,200	51	Negligible	95	Negligible	
Refractory							
Noncast	75	55,000	b/	4,100	64	1,500	9.0
Electric cast	112	6,000	b/	684	77	150	1.0
Chemical processing							
Dichromate	15	55,400	b/	835	90	84	0.6
Other chemicals						22	
Steel and alloys							
Chromium steels	12	172,000	b/	2,100	78	472	2.9
Cast iron	38	4,500	b/	171	99	1.8	0
Super alloys and alloys	12	11,000	b/	136	78	30	0.2
General steelmaking	NA[c]	NA	NA	NA	NA	91	0.6
Inadvertent sources							
Coal combustion	NA	30,700,000	0.026	7,900	82	1,420	8.6
Oil combustion	NA	261,000	0.13	336	0	336	2.0
Cement production	NA	848,000	0.03	NA	NA	254	1.5
Incineration	NA	845,000	0.017	NA	NA	143	0.9
Asbestos	NA	6,000	0.15	9.1	99	0	
Total				36,100	54	16,500	

Adapted from GCA Corporation (1973), Table 2, p. 12.

[a] Intermediate value.

[b] Emission factor multiplier equal to tons of chromium processed or handled annually.

[c] NA - not applicable.

Table 4.15. Chromium Emission Sources

Source	Chromium Emission: Amount (metric tons)	Chromium Emission: Percent of This Pollutant
Asbestos minimg	7.3	0.07
Kraft pulp mill recovery furnace	Negligible	Negligible
Sulfite pulp mill	Negligible	Negligible
Primary chromium production	3,800	34.98
Asbestos products	Negligible	Negligible
Refractory brick production	6.3	0.06
Installation of asbestos material	Negligible	Negligible
Spray-on fireproofing	Negligible	Negligible
Use of insulating cement	Negligible	Negligible
Powerplant boilers		
Pulverized coal	5,100	46.40
Stoker coal	580	5.33
Cyclone coal	170	1.60
All oil	20	0.18
Industrial boilers		
Pulverized coal	220	2.06
Stoker coal	780	7.20
Cyclone coal	110	1.02
All oil	15	0.14
Residential/commercial boilers		
Coal	70	0.64
Oil	34	0.32
Total	10,900	

From Goldberg (1973), Appendix A, p. 107.

Chromic chloride, after an exposure time of 20 hours at a concentration of 5.48×10^{-3} M, caused a 50% reduction in the viability of rabbit alveolar macrophages (Waters et al. 1975). Waters et al. also report that 20-hour exposure to 8.57×10^{-3} M results in lysis of 50% of the cells. Incubation of B lymphocytes from mice with from 10^{-7} to 10^{-3} M chromic chloride for a period of 72 hours reduced the fraction of surviving cells compared to control cultures (Shenker et al. 1977). The same concentration range (10^{-7} to 10^{-3} m) was found to stimulate incorporation of thymidine and alanine into high molecular weight products during the final 18 hours of the exposure regime. Cultured hamster fibroblasts are killed by 24-hour exposure to potassium dichromate (Levis et al. 1978). The survival curve as a function of dichromate concentration exhibits a shoulder followed by exponential killing at concentrations above 7×10^{-6} M.

Chromium compounds also affect nonmammalian cells. Growth of yeast colonies on media containing 0.961 mM calcium chromate results in a three-to five-fold increase over the spontaneous frequency in the number of respiration-deficient colonies (Egilsson et al. 1979). Eighteen-hour exposure of yeast to 10-mM chromous chloride in 1% glucose solution results in 0.01% survival (Putrament et al. 1977). After three hours, DNA synthesis is decreased 84% and after six hours, 92%. Both potassium chromate and potassium dichromate cause increased lethality in strains of bacteria that lack DNA repair capability compared to proficient strains (Nishioka 1975). Petrilli and DeFlora (1977) have studied the toxic and mutagenic effects of hexavalent chromium compounds in Salmonella typhimurium. In a strain in which most mutational events involve an error-prone repair system (i.e., TA100), revertants were found to occur at frequencies proportional to the square root of the chromium concentrations, there was a sharp decrease in the number of revertants.

Chromium effects can also be demonstrated in cell-free systems. Sirover and Loeb (1976) found that 0.64-mM chromous chloride triples the frequency of misincorporation nucleotides in in vitro

DNA synthesis. Chromic acid at a concentration of 16 mM also triples the frequency of misincorporation. In a similar experiment, Tkeshelashvili et al. (1980) found that both chromic acid and chromic chloride, over the concentration range 0-5 mM, monotonically increased misincorporation. Aitio et al. (1978) found that 0.25-mM chromium potassium sulfate had no effect on epoxide hydratase and glutathione S-transferase, two key enzymes in the detoxification of xenobiotic compounds, such as polynuclear aromatic hydrocarbons.

These effect levels are summarized in Figure 4.25.

4.3.2.5 Mercury

Joensu (1971) reports that 3,000 tons of mercury (Hg) are mobilized annually by coal combustion on a global basis, with 1,800 tons attributed to the U.S. The largest single source of mercury released to the atmosphere in the U.S. is fossil-fuel-burning powerplants; ~22% of the total atmospheric emissions from industrial sources come from powerplants (Oglesby 1975). Approximately 90-97% of the mercury concentration in coal (0.02-1.91 μg/g) and oil (0.002-30 μg/g) is released upon combustion in the flue gas (Oglesby 1975; Billings et al. 1973; Anderson and Smith 1977), of which 92-99% is in the form of elemental mercury vapor (Lindberg 1980). Contrary to the behavior of other particulate-associated metals, mercury vapor does not appreciably collect on particulates and thus is in a form conducive to long-range transport and removal from the atmosphere by precipitation scavenging (Natusch 1978; Lindberg 1980). The remaining mercury is in the slag (Bolton et al. 1974) and in the fly ash, which may have up to 22 ppm mercury (Cowherd 1975; Oglesby 1975). Bolton et al. (1974) also have determined that the degree of enhancement in gaseous samples is much larger for coals with relatively small amounts of mercury (0.05-0.06 ppm) than for coals with mercury concentrations (0.19-0.22 ppm). Depletion of the high stack concentrations by fallout and diffusion is rapid, and resulting ground level concentrations for the least favorable meteorological conditions have been estimated to be between 0.004 and 0.1 $\mu g/m^3$ by Bolton et al. (1974). Vaughn et al. (1975) predicted concentrations of about 0.0012 $\mu g/m^3$. These values are between ten and a few hundred times lower than the maximum level (1 $\mu g/m^3$) currently recommended by USEPA (Cowherd et al. 1975). However, Lindberg (1980) measured mercury concentrations in the gas plume at various distances from a coal-fired powerplant and found 1.7 $\mu g/m^3$ Hg vapor at 0.25 km from the plant decreasing to 0.02 $\mu g/m^3$ at 22 km (background of 0.012-0.020 $\mu g/m^3$). Thus, measured values may exceed predicted values and USEPA-recommended standards.

In examining the potential health effects of mercury released into the atmosphere, ambient airborne concentrations and related fallout and washout with subsequent transfer to man by water and food sources must be considered. The chemical form of the mercury plays a very important role in transport by water and food and in its final effect on man. In the natural background atmosphere, mercury is present as both vapor and particulates at concentrations of 1 $\mu g/m^3$, but may be from 20-200 $\mu g/m^3$ in areas with high mercury soil levels (Beliles 1975). Ingestion of metallic mercury is not ordinarily toxic to man, but inhalation of mercury vapor can be injurious. From a toxicological point of view mercury compounds are separable into two classes: inorganic, in which mercury is present either as free metal or in ionic form, such as a mercurous or mercuric salt (e.g., mercurous chloride or mercuric nitrate); and organic, in which the mercury is bound covalently to at least one carbon atom (e.g., dimethyl mercury or phenylmercuric acetate). Organic mercury compounds are considerably more toxic than inorganic ones. The alkyl compounds, including methyl, are particularly toxic. However, neither air quality standards nor water quality standards are based on distinction of compounds (Hausknecht and Ziskind 1976).

The maximum allowable concentration for ambient air set by the Environmental Protection Agency (USEPA) is 1 $\mu g/m^3$ for mercury. The USEPA is formulating an air quality criterion (AQC), which will be the legal basis for providing assistance to the states in taking technological and political action to protect the public health. Occupational exposures are regulated at 50 $\mu g/m^3$ as a timeweighted average for an eight-hour day, five days per week (NIOSH 1973b). For cinnabar smelting and use in chlor-alkali production there is a National Emission Standard of 2300 g or 5.0 lb in a 24-hour period. The primary exposure route of man to atmospheric sources is the respiratory tract. Dose effects of inhalation exposure and dietary ingestion have received considerable attention and have been thoroughly reviewed by U.S. Congress (1970); Goldwater (1971); Friberg et al. (1971); Joselow (1972); NIOSH (1973b); Trachtenberg (1974); Hausknecht and Ziskind (1976); and Petering and Tepper (1976).

Relatively few data are available to define a dose-effect relationship for chronic inhalation of organic mercury compounds, but a significant amount of data are available from chronic occupational exposures to mercury vapor and dust carrying unspecified mercury compounds which are primarily inorganic (Fig. 4.30) (Hausknecht and Ziskind 1976).

Alkyl mercurial compounds attack the brain and in one instance, inhalation of diethyl mercury at an estimated level of 1 mg/m^3 for several months, caused two fatalities (Hill 1943). A study of 20 workers exposed to monthly average levels of mercury in air of 0.03-0.1 mg/m^3 caused by dust of ethyl mercury chloride or phosphate as well as solvent solutions of ethyl and phenyl mercury acetate resulted in no significant objective findings (Dinman et al. 1958).

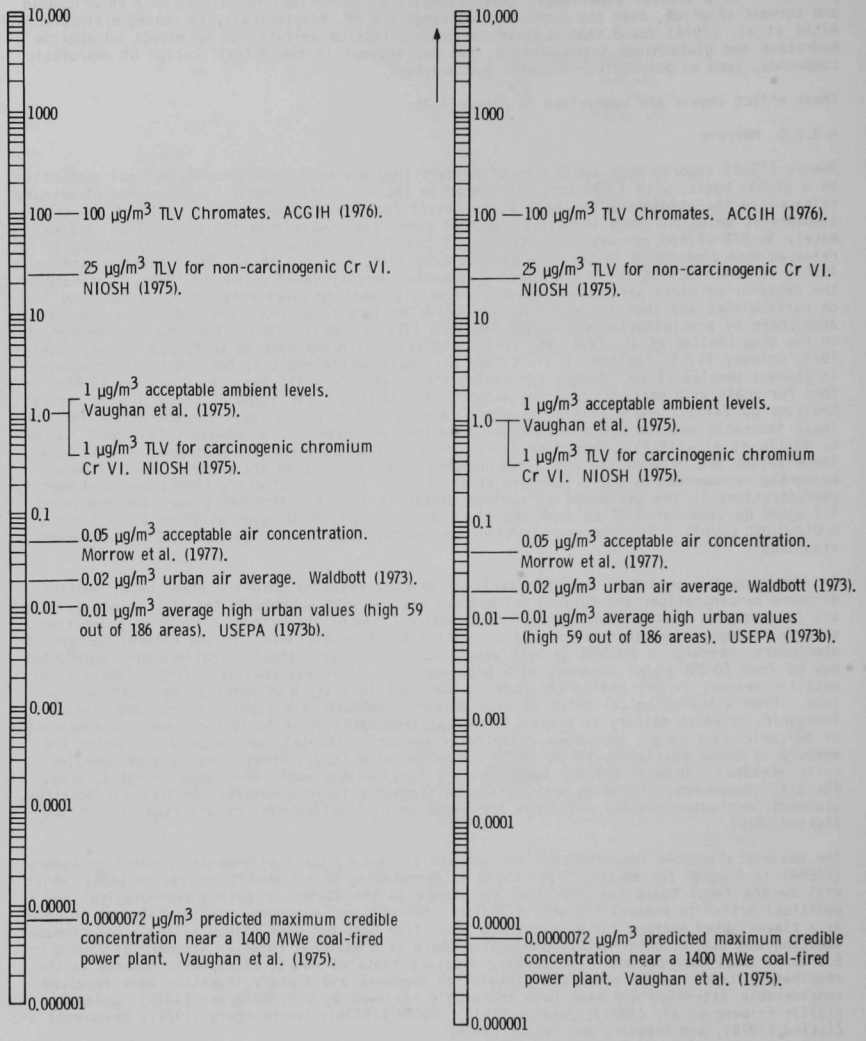


Fig. 4.29. Levels and Some Health Effects of Chromium (Cr)

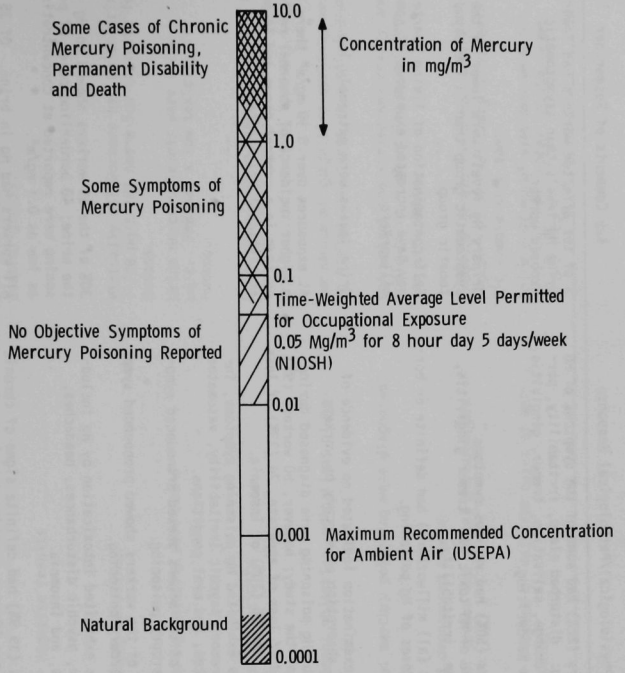


Fig. 4.30. Occupational Data (Chronic) for Inhalation of Mercury Vapor and Inorganic Compounds. Redrawn from Hausknecht and Ziskind (1976).

Since most of the mercury released by coal-fired plants is in the form of elemental vapor and because of its lifetime in the atmosphere is short and may preclude substantial chemical conversion, organic mercurials are not expected to be an inhalation health risk relevant to powerplants (Hausknecht and Ziskind 1976). The relationships between exposure to inorganic mercury and symptoms have been examined in depth, providing the basis for the current industrial maximum air concentration ranges. Exposure data for inhalation of inorganic mercury, primarily through epidemiologic examination, are summarized in Table 4.16 (Hausknecht and Ziskind 1976). In one comprehensive study by Smith et al. (1970), 567 workers who had been exposed to mercury (generally, 90% as mercury vapor) were examined. A clear dose response to mercury exposure, including nervous system changes indicative of mercury poisoning, is shown in Figure 4.31. Although some effects were noted at the smallest values used (10-50 $\mu g/m^3$), Smith et al. (1970) concluded that no significant signs or symptoms were present in persons exposed to <10 $\mu g/m^3$ there have been incidents of relatively mild mercury intoxication, and expressed some concern about the adequacy of the safety factor of a TLV at this level.

Ingestion of elemental mercury or inorganic mercury compounds does not result in significant health effects, but organic mercury ingestion can result in death, nervous system damage, and other severe symptoms (Petering and Tepper 1976).

Table 4.16. Summary of Exposure Data for Inhalation of Inorganic Mercury

Industry	Number Exposed	Estimated Dose Level (mg/m^3) and Duration	Physiological/Pathological Response	Key Comments of Researcher
Cinnabar and native mercury mines	74 miners and smelters (males)	0.16 to 4.84 of dust and vapor with vapor at 0.1 to 2.0 in the mine and 0 to 2.0 in the smelter	16 workers (22%) had some of the symptoms of HG poisoning: disturbed sleep, irritability, personality change, salivation, tremor, gingivitis, tremulous handwriting.	3 of the affected workers had lower urine Hg levels than asymptomatic exposed worker.
Open pit cinnabar mine	30 miners (males)	0.1 to > 2.0 of vapor (dust was not measured)	15 workers (50%) had various symptoms suggestive of HG toxicity: tremor gingivitis, salivation, irritability.	Urinary Hg levels were lower in the symptomatic group than in the asymptomatic group.
Mining and milling	96 workers	0.3 to $\leq$ 1.2 for an average of 8 months for those which manifested symptoms	3 workers (all millworkers) had definite or borderline doses of Hg poisoning.	Self-contamination of living quarters may have increased exposures of some millworkers.
Chlor-alkali	--	0.08 to 0.13	Physical examination indicated no evidence of dangerous absorption of mercury by workers.	Urine levels were extremely low.
Chlor-alkali	567 workers in 21 plants (6-14 years in industry)	0.01 to 0.27 of vapor; total airborne concentration unknown	No cases of Hg poisoning were diagnosed during the year of the study; however, 50 workers (9%) complained of loss of appetite, 74 (13%) of loss of weight, and 56 (10%) of insomnia.	At exposures over 0.10 mg/m^3 there was a higher incidence of abnormal reflexes than in a control group and it was statistically significant.
Hatter's fur-cutting	529 workers	0.06-0.72 total	43 workers exhibited Hg poisoning symptoms, including tremor, psychic irritability, vasomotor disturbances, and oral conditions.	--
Mercury mine	130 miners	1.2-5.9	One-third of the miners showed pronounced symptoms of mercury poisoning.	--
Mercury smelter	59 workers	0.25-0.85	One-third of the workers showed pronounced symptoms of mercury poisoning.	--
Hatter's fur-cutting	534 workers	Up to 0.5 total	59 workers exhibited intoxication by Hg including tremor, psychic disturbances, headaches, drowsiness, and insomnia.	30% of the 59 workers showed no Hg in the urine; 40 borderline cases of poisoning were reported at concentrations as low as 0.1 mg/m^3.
Fur-felt	213 workers	<0.1-0.81 total	85 workers (39.9%) had definite signs of chronic Hg poisoning; 58 were considered to be borderline cases.	All workers had Hg in urine. Of 35 workers exposed to < 0.1 mg/m^3, 4 had signs or symptoms of Hg poisoning and 10 were borderline cases.
Hatter's felt-cutting	1173 workers	0.5-2.0	300 cases of Hg poisoning with one-third of the cases resulting in permanent disability.	Some cases were reported at levels below 0.5 mg/m^3, but no cases were observed below 0.1 mg/m^3.

Table 4.16. (concluded)

Industry (Reference No.)	Number Exposed	Estimated Dose Level (mg/m^3) and Duration	Physiological/Pathological Response	Key Comments of Researcher
Felt hatter's	70 females	0.25-1.0	Two-thirds showed pronounced symptoms of Hg poisoning	No significant differences between the values of blood elements and hemaglobin levels between the exposed and control groups.
Varied	--	0.032-0.40 (variety of inorganic compounds with metallic Hg)	Clinically negative results	--
Varied	23 workers	Up to 0.08 Hg vapor with PMB on skin	No signs of Hg poisoning	Virtually all workers had Hg in urine. 1-788 µg/ℓ PMB has a low toxicity for humans.
Varied	21 workers	0.1 total which was essentially all vapor with PMB on skin	No signs of Hg toxicity were found	Urine mercury levels were 0-240 µg/ℓ. PMB has a low toxicity for humans.
Varied	23 workers	0.05-0.10 at some locations; no detectable Hg at others; PMA on skin	No signs of Hg toxicity	Urine levels were below 150 µg/ℓ. PMA has a low toxicity for humans.
Varied	20 workers	0.01-0.12 ethylmercuric acetate and PMB on skin (5½ years)	No significant objective indications of Hg poisoning	Incidence of subjective symptoms were no higher than those in control groups.
Meter repair	161 workers	0.05-0.3	26 cases of mercury poisoning were diagnosed	Concentrations varied seasonally and could be as high as 1-6 mg/m^3 over the work desks.
Chlorine plant	91 workers	0.1-1	7 cases of pronounced tremor	--
Varied	58 workers	0.01-0.06	15 cases of tremor and mental disturbances	Exposure levels for other workers were higher and several cases of hyperchromic anemia were seen.
Varied		0.08-0.68	6 cases or suspect or definite cases of mercury poisoning	Below 0.02 mg/m^3 symptoms were not seen, but did appear in the range 0.2-0.3 mg/m^3.

From Hausknecht and Ziskind (1976).

PMB is phenylmercuric benzoate.

PMA is phenylmercuric acetate.

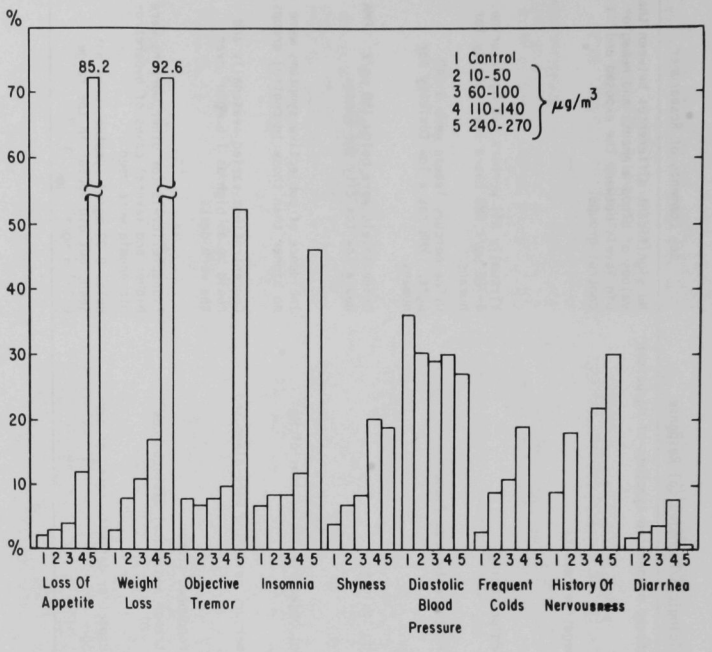


Fig. 4.31. Percentage Prevalence of Certain Signs and Symptoms Among Workers Exposed to Mercury in Relation to Degree of Exposure. Redrawn from Smith et al. (1970).

Organic mercury compounds may occur in the aquatic environment due to biotransformation of inorganic mercury compounds by bacteria in the sediments. Industrial mercury chloride dumped in Minimata Bay and other areas of Japan was biologically converted to methyl mercury and concentrated through the food chain to fish which were eaten by humans. "Minimata Disease" has caused many deaths and many severe neurological disorders. There are 784 known patients, of which 103 have died; about 3,000 more persons are suspected of suffering from this disease. Indications are that the standard accepted 70-day biological half-life usually accepted for mercury is not true in this instance, since many individuals begin to exhibit symptoms many years after exposure. Fetuses appear to be particularly susceptible, and infants may be born with severe neurological disease from mothers who show no effects of mercury exposure. There does not appear to be a real threshold limit for exposure to methyl mercury since any amount taken seems to cause some cell damage. Dose levels that cause observable symptoms are not known at present (Harada 1978).

The fallout of mercury in the vicinity of a large coal-fired plant can lead to increased concentrations in nearby waterways, causing the resulting concentrations in edible fish and shellfish to be of concern with respect to human health in much the same ways as with Minimata Bay. Methylating organisms are known to exist in bottom sediments of waterways and the food chains leading from ambient water concentrations to the higher trophic levels result in bioamplification of the mercury concentration. In a study of a recent water impoundment (Lake Powell) that is especially vulnerable to mercury pollution because of reduced sediment transport, it was estimated that an additional large coal-fired plant (Kaiparowits) in the vicinity could raise the mercury content of edible fish above the regulatory level (500 ppb) set by the U.S. Food and Drug Administration (Standiford et al. 1973). While these results are preliminary, the impact of large coal-fired plants on the mercury content of edible fish and shellfish in nearby waterways must be taken into consideration until definitive measurements and analysis of the mercury transport and chemical reactions after leaving the stack are available.

Mercury exposure is toxic to animals and also induces many subcellular changes (Fowler 1978). Liver and brain cells of animals exposed to 10 mg/kg methyl mercury exhibit elevated rates of DNA, RNA, and protein synthesis. In contrast, renal cells of these animals exhibited decreased rates of DNA and protein synthesis, but elevated RNA synthetic rates. In another experiment, prolonged exposure to methyl mercury resulted in morphological changes of mitochondria in liver and kidney cells. Such changes were associated with changes in mitochondrial enzyme functions, including monoamine oxidase, cytochrome oxidase, and δ-aminolevulinic acid synthetase. Mercury derived from methyl mercury accumulates in lysosomes where it decreases β-glucuronidase activity and increases acid phosphatase activity. Injected doses of five to ten mg/kg of methyl mercury result in about 50% reduction of hepatic microsomal enzyme activities (Fowler 1978). Rozalski and Wierzbicki (1979) have found that after injections of 20 mg/kg of mercuric chloride into rats, there was an accumulation of mercury in the nuclei of liver and kidney cells. About 10% of the mercury recovered was in the nucleus and of that more than half was bound to chromatin.

The toxic dose of mercury chloride (HgCl) for human leukocytes in culture was determined to be 3.0×10^{-8} M (Paton and Allison 1972). While studying rat alveolar macrophages, Castranova et al. (1980) found that half maximal inhibition of total oxygen consumption and level of reactive oxygen species occurred at mercuric ion concentrations of 2.5×10^{-5} and 2.3×10^{-6} M, respectively. Ashida (1965) reviewed the literature concerning metal toxicity in fungal species. In nine out of ten species where the experiments had been done, the fungi could adapt to growth in the presence of mercuric ion. Nishioka (1975) tested four mercury-containing compounds for differential toxicity in bacterial strains with and without DNA repair capability. Both mercurous and mercuric chloride showed no difference in toxic response, whereas the two compounds in which the mercury atom is covalently bonded to carbon--phenyl mercury acetate and methyl mercury chloride--do show higher toxicity in repair-deficient strain. This suggests that the latter compounds attack DNA or modify its metabolism.

In a cell-free system, Aitio et al. (1978) determined the effect of mercuric acetate on various microsomal enzymes. They found that 2×10^{-6} to 5×10^{-4} M Hg inhibited mixed-function oxidase activity and that 4.3×10^{-4} M Hg inhibited both epoxide hydratase and glutathione S-transferase activities. In another system, Korman et al. (1978) found that 2.5×10^{-5} M HgCl reduced *Micrococcus luteus* DNA polymerase I activity *in vitro* by 50%.

These effect levels are summarized in Figure 4.32.

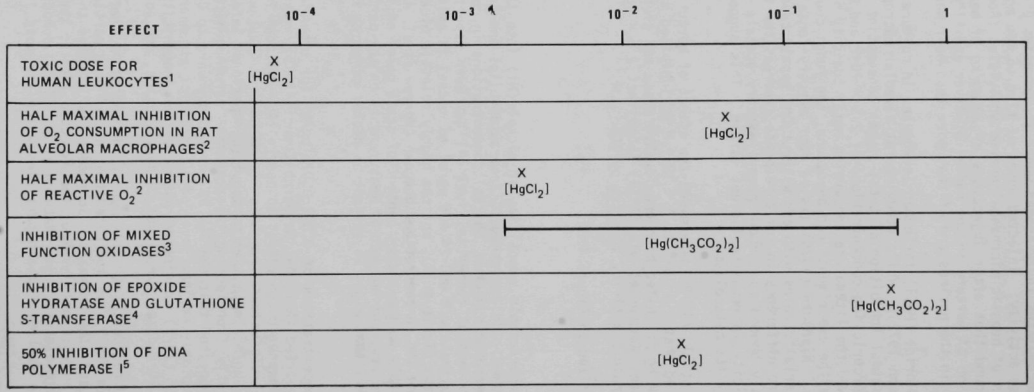
4.3.2.6 Nickel

Coal-fired powerplants emit appreciable quantities of nickel (Ni) into the atmosphere, but emissions from space heating fuel oils and exhausts from automobiles and trucks also are implicated as major sources of atmospheric nickel. Nickel concentrations in coals vary from 5.3 to 19.4 ppm, while the nickel content of commercial fuel oil has been reported as ranging from nearly zero to 20 ppm. Approximately 8,800-24,400 μg/m^3 of nickel is emitted from coal-fired powerplants in collected fly ash (Table 4.17), but the highly volatile nickel carbonyl ($Ni(CO)_4$) is absent from the fly ash and its concentrations unknown (Morrow et al. 1977; Van Hook 1979). There are no known data available on the nickel content of stack gases of space heating plants. Diesel-engine exhaust data are few, but one set of measurements on the particulate phase, which also does not include any nickel that might be present as nickel carbonyl in the vapor phase, is given in Table 4.18.

Average atmospheric nickel concentrations of 0.017-0.025 μg/m^3 for urban air and 0.006 μg/m^3 for nonurban air have been reported (National Research Council 1975). Vaughn et al. (1975) predict a maximum credible atmospheric concentration near a 1400-MWe coal-fired powerplant to be 4.8×10^{-4} μg/m^3. Threshold limit values have been recommended at 1000 μg/m^3 for nickel and nickel soluble compounds and 7 μg/m^3 for nickel carbonyl (Occupational Safety and Health Administration 1971), and more recently (considering the potential carcinogenicity), the National Research Council (1975) has suggested that the TLV be revised downward to 0.1 μg/m^3. Vaughan et al. (1975) recommended an acceptable ambient level of 10 μg/m^3 for air near a coal-fired powerplant; however, Morrow et al. (1977) considered potential carcinogenicity and recommended 0.01 μg/m^3 for nickel and 1×10^{-6} μg/m^3 for nickel carbonyl, oxide, and sulfide.

Nickel may be inhaled by urban residents at ~2-14 μg/day, depending on time and location (National Research Council 1975). Graham (1975) has indicated that nickel as a particulate has deleterious health effects in both humans and other mammals. Schroeder (1971) notes that the inhaled nickel actually retained within the body is only ~75% of the expected intake; the remainder would be expired, depending upon the size distribution. Natusch et al. (1974) demonstrate that nickel is preferentially adsorbed on the smaller fly ash particles which are most likely to reach the lungs.

The chemical species of nickel affects the toxicity in several ways. Nickel carbonyl is generally accepted as being a potential carcinogens, and nickel oxide and sulfide are considered to be possible carcinogens (Morrow et al. 1977; Van Hook 1979; Natusch 1978). Nickel carbonyl is the most toxic of all nickel compounds to man, causing death after exposure of 30 μg/g for 30 minutes (Beliles 1975). It is formed when hot CO is passed over nickel; both of these are waste products



1. Paton and Allison (1972)
2. Castranova et al. (1980)
3. Aitio et al. (1978)
4. Aitio et al. (1978)
5. Korman et al. (1980)

Fig. 4.32. Concentrations of Mercury Causing Cellular Effects (mM)

Table 4.17. Nickel Emissions from Coal-Fired Powerplants[a]

Powerplant Boiler Type	Nickel in Collected Fly Ash	
	μg/SCF[b]	μg/m³
Vertical	250[c]	8,800
Corner	130[c]	4,600
Front-wall	170[d]	6,000
Spreader-stoker	350[e]	12,400
Cyclone-fired unit	510[d]	18,000
Horizontally opposed	690[e]	24,400

From National Research Council (1975).

[a]Derived from Cuffe and Gerstle.

[b]SCF = standard cubic foot.

[c]Fly-ash collector is cyclone separator followed by electrostatic precipitator.

[d]Fly-ash collector is electrostatic precipitator.

[e]Fly-ash collector is cyclone separator.

Table 4.18. Nickel Concentrations in Diesel-Engine Exhaust

Substance	Nickel Concentration (μg/g of particles)
Bulk diesel fuel[a]	2
Exhaust-valve coke	10
Particulate sample[b]	10,000 (0.65 μg/min)
Particulate sample[c]	1,000
Particulate sample[d]	500

From National Research Council (1975).

[a]N.Y. Central Spec. 1370-C, Grade 2.

[b]No load at 1,400 rpm.

[c]No load at 1,800 rpm.

[d]Half load at 1,800 rpm.

of coal-fired plants (Heit 1977). Epidemiological studies have correlated nickel carbonyl to increased risk of lung and nasal cancers (see Sunderman 1973, 1976, 1977; National Research Council 1975; Fishbein 1976; International Agency for Research on Cancer 1976). More than 447 cases of lung cancer and 143 nasal cancer have occured among nickel workers in the United States, USSR, Canada, Germany, Norway, England, France, and Japan (Sunderman 1976). Sunderman (1976) reports no epidemiological studies of mortality among groups of workers who were not employed in nickel refineries, but who were chronically exposed to inhalation of nickel compounds; he does reference three case studies and provide one case study of worker exposure in nickel-plating and grinding operations. Inhalation exposure studies with experimental animals have confirmed these findings for nickel carbonyl and for nickel oxide and sulfide, as shown in Table 4.19 (Sunderman 1976). No values were found for concentrations presenting potential cancer risks.

Human body burdens of nickel are estimated at about 10 mg for a 70-kg person (Schroeder 1965). Daily ingestion rates are about 300-600 μg from food and water; however, most of this passes through the gastrointestinal tract unabsorbed (Schroeder et al. 1962; National Research Council 1975).

Table 4.19. Experimental Models of Nickel Carcinogenesis

Author[a]	Animal(s)	Compounds	Route(s)	Tumor(s)
Heuper	Rat, rabbit	Ni dust	Intraosseous, intrapleural	Sarcoma
Heuper	Guinea pig	Ni dust	Inhalation	Anaplastic, adenocarcinoma (lung)
Sunderman et al.	Rat	$Ni(CO)_4$	Inhalation	Epidermoid, anaplastic, adenocarcinoma (lung)
Gilman	Rat, Mouse	Ni_3S_2 dust NiO dust	Intramuscular	Rhabdomyosarcoma
Heath et al.	Rat	Ni dust	Intramuscular	Rhabdomyosarcoma
Furst et al.	Rat, Hamster	Nickelocene	Intramuscular	Sarcoma
J.P.W. Gilman	Cat	Ni_3S_2 discs	Sinus implants	Epidermoid, adenocarcinoma; sarcoma
Lau et al.	Rat	$Ni(CO)_4$	Intraveneous	Carcinomas, sarcoma
Furst and Cassetta	Rat	Ni dust	Intrathoracic, intraperitoneal	Mesothelioma
Ottolenghi et al.	Rat	Ni_3S_2 dust	Inhalation	Epidermoid, adenocarcinoma (lung)
Sosinski	Rat	Ni_2O_3 dust	Intracerebral	Sarcoma, meningioma
Stoner et al.	Mouse	$Ni(C_2H_3O_2)$	Intraperitoneal	Adenocarcinoma (lung)
Jasmin and Riopelle	Rat	Ni_3S_2 dust	Intrarenal	Adenocarcinoma
Sunderman et al.	Hamster	Ni_3S_2 dust	Intramuscular	Sarcoma
Damjanov et al.	Rat	Ni_3S_2 dust	Intratesticular	Sarcoma

From Sunderman (1976).

[a]Authors cited are in the source and are omitted in this report.

At present the chemical nature of nickel from coal-fired powerplants is unknown. Carcinogenesis of some nickel compounds has been clearly demonstrated; however, there is no evidence demonstrating the presence or absence of these compounds in coal-fired powerplant effluents. Morrow et al. (1977) recommends 1×10^{-6} $\mu g/m^3$ as an acceptable air concentration, but other groups such as the National Research Council have set values at 0.1 $\mu g/m^3$, which is much higher than the Morrow recommendation. Since the chemical nature of nickel from coal combustion is unknown, we must assume that all of it is in the carcinogenic form. If so, according to the Vaughan et al. (1975) estimate of 0.00048 $\mu g/m^3$, it would be roughly 200 times below the National Research Council (1975) recommended threshold limit value. If the Morrow et al. (1977) acceptable level is used, then nickel from coal combustion would exceed the acceptable amount by 480-fold. Some of the above exposure and health effects data are shown in Figure 4.33.

Some people have skin reactions, such as dermatitis, to direct contact with elemental nickel used in jewelry and coins. As much as 5% of eczema cases may be due to nickel exposures. Effects of exposure to nickel carbonyl range from headaches to death.

Nickel has demonstrable genotoxic effects. There are several reports of cancer in experimental animals following exposure to nickel (Sunderman 1978). This metal is toxic to mammalian cells, toxic and mutagenic to microbial cells, and inhibitory to enzymatic reactions in vitro.

Waters et al. (1975) studied the toxic effects of nickel upon rabbit alveolar macrophages. Twenty-hour exposure to nickel-chloride concentrations ranging from 1 to 20 mM result in reduced cell viability. Exposure from 3 to 20 mM results in cell lysis and reduced cell number. Rat alveolar macrophages are also affected by nickel (Castranova et al. 1980). Fifteen-minute exposure to 0.01 M nickel chloride results in 73% inhibition of oxygen consumption, while exposure to 0.0039 M solution causes 36% inhibition.

Some studies of nickel have used lower eukaryotes and prokaryotes. Egilsson et al. (1979) found that 15 mM nickel chloride in the growth medium resulted in a three- to five-fold increase in the frequency of respiratory deficient yeast colonies. It has been shown that 18-hour exposure to 10 mM nickel chloride inhibits yeast growth but not in the presence of equimolar concentrations of magnesium chloride (Putrament et al. 1977). Six-hour exposure inhibited nuclear DNA synthesis

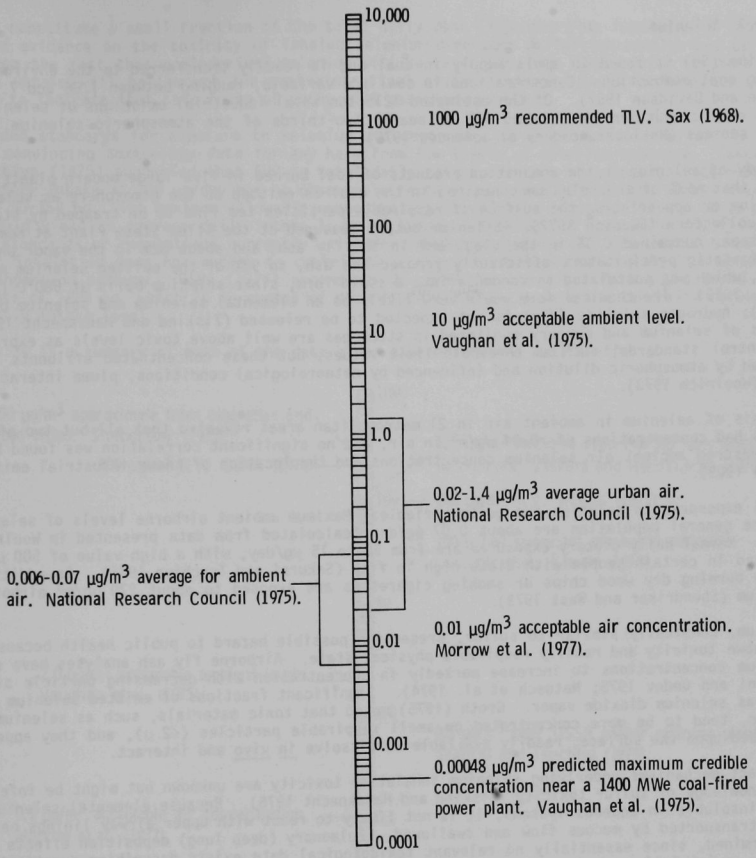


Fig. 4.33. Levels and Effects of Nickel (Ni)

by 83%, but mitochondrial DNA synthesis by only 27%. Bacterial strains lacking DNA repair capability are no more easily killed by nickel chloride than are repair-proficient strains (Nishioka 1975).

Effects of nickel also are evident in cell-free systems. There was a two-fold increase over background in misincorporation of nucleotides in an in vitro DNA synthesis experiment when 8 mM nickel chloride was present in the incubation mixture (Sirover and Loeb 1976). The enzymatic activity of DNA polymerase I from Micrococcus luteus is reduced 50% when 55 mM nickel chloride is present in vitro (Korman et al. 1978). In contrast, 250 mM nickel sulfate had no effect on epoxide hydratase or glutathione S-transferase activities in vitro, both of which are enzymes involved in detoxification of polycyclic organic molecules (Aitio et al. 1978).

These effect levels are summarized in Figures 4.20 through 4.23.

4.3.2.7 Selenium

Selenium (Se) is found in ample supply in coal and is readily transferred to the environment during coal combustion. Concentrations in coal are variable, ranging between 1.96 and 7.38 ppm (Lakin and Davidson 1967). Of the estimated 1215-ton total industrial emissions of selenium for 1970, coal combustion contributed 62% or nearly two-thirds of the atmospheric selenium from these sources (National Academy of Sciences 1975).

A study of selenium in the combustion products of coal burned in five large modern plants indicates that ~53% of the selenium contained in the coal is emitted to the atmosphere as volatilized selenium or appearing on the surface of respirable particles too fine to be trapped by standard dust collectors (Swanson 1972). Selenium output measured at the Allen Steam Plant at Memphis, Tennessee, contained 0.3% in the slag, 68% in the fly ash, and about 32% in the vapor phase. Electrostatic precipitators efficiently removed the ash, so 93% of the emitted selenium was as vapor, which was postulated to condense into a solid form, since selenium boils at 680°C (Andren et al. 1975). The chemical form would very likely be an elemental selenium and selenium dioxide (SeO_2); hydrogen selenide (HSe) is not expected to be released (Ziskind and Hausknecht 1976). Levels of selenium and mercury monitored in stack gas are well above toxic levels as expressed by control standards, such as threshold limit values, but these concentrated effluents are diluted by atmospheric dilution and influenced by meteorological conditions, plume interactions, etc. (Woolrich 1973).

Analysis of selenium in ambient air in 21 metropolitan areas revealed that all but two of the cities had concentrations of >0.04 μg/m^3 in air, and no significant correlation was found between the measured ambient air selenium concentrations and the location of known industrial emitters (Stahl 1969).

Actual exposures to selenium are quite variable. Maximum ambient airborne levels of selenium for the general population are about 0.07 μg/day (calculated from data presented in Woolrich 1973). Normal daily dietary exposures are from 50 to 15 μg/day, with a high value of 500 μg/day observed in certain people with diets high in fish (Sakurai and Tsuchiya 1975). In addition, people burning dry wood chips or smoking cigarettes are exposed to about 270 μg/m^3 airborne selenium (Shendrikar and West 1973).

Selenium, chemically similar to sulfur, presents a possible hazard to public health because of its known toxicity and readily respirable physical state. Airborne fly ash analyses have shown selenium concentrations to increase markedly in concentration with decreasing particle size (Ragaini and Ondov 1975; Natusch et al. 1974). Significant fractions of emitted selenium also exist as selenium dioxide vapor. Groth (1975) noted that toxic materials, such as selenium and arsenic, tend to be more concentrated on small respirable particles (<2 μ), and they appear localized upon the surface, readily available to dissolve in vivo and interact.

The exact mechanisms underlying selenium inhalation toxicity are unknown but might be inferred from knowledge of sulfur toxicity (Ziskind and Hansknecht 1976). Because elemental selenium is quite insoluble in aqueous systems, it is not likely to react with upper airway linings before being transported by mucous flow and swallowed. Pulmonary (deep lung) deposition effects are undetermined, since essentially no relevant toxicological data exists describing quantitative effects on inhalation of gaseous or particulate selenium compounds important to coal combustion. In the National Academy of Science's comprehensive review of selenium (1976), of 845 citations, none was directly applicable to inhalation studies (Ziskind and Hausknecht 1976; Vesar 1976). Unpublished data were reported by Ziskind and Hausknecht (1976) from a study with animals exposed to 12 mg/m^3 bismuth telluride doped with 1% or less selenium (120 μg/m^3), and no acute toxicity or possible cancer precursor lesions were found. Because of possible inadequacies in the research design, these results may not be conclusive.

Pulmonary deposition may be important since airway retention could last for months. In the same manner as sulfur dioxide, selenium dioxide directly irritates the airway epithelium, probably causing reflex bronchoconstriction, and probably can readily infiltrate across tissue layers and be taken up into the blood flow. Harmful effects might be caused by the organism's substitution of selenium for sulfur in certain enzyme systems, with the selenium compounds being more reactive and less stable than sulfur compounds (Stadtman 1974).

Human toxicity has been well reviewed: National Academy of Sciences (1975); Rosenfeld and Beath (1964); Muth (1967); Scott (1973); Sakurai and Tsuchiya (1975); Allaway (1973); Diplock (1976); and Shapiro (1973). Selenium is readily absorbed through the small intestine. Up to 50% of ingested selenium is excreted in the urine, and the portion retained in the body accumulates primarily in the liver and the kidneys (Lo and Sandi 1980). Chronic industrial exposures to relatively high levels of selenium compounds lead to nasal bleeding, loss of smell, garlic breath, dermatitis, headache, and intense irritation of eyes and nasopharyngeal passages; however, long-term latent effects are known. One 13-year study of industrially exposed workers showed no long-term effects (Cooper 1967). A rough estimate of the lethal dose for humans can be made by using an average of animal LD_{50} data presented in National Academy of Sciences (1975) of about 3 mg/kg body weight; for a 70 kg human this would be about 210 mg. Daily inhalation doses of

selenium constitute a small fraction of the total daily dose (ingested plus inhaled). A lack of published evidence on the toxicity of inhaled selenium precludes definitive conclusions to be drawn from the fact that coal-derived selenium will be taken into the lungs, while the bulk of selenium uptake will be through the gastrointestinal tract. However, there is likewise no evidence to indicate that this route of entry will cause any problems.

Recommended standards for exposure to selenium are controversial at present, due primarily to lack of convincing toxicology data for any harm from low levels of selenium exposure. Sakurai and Tsuchiya (1975) suggested that 500 μg total daily selenium intake be adopted, since people with dietary intake at 500 μg/day had no observable toxic effects even though the normal average range is 50-150 μg. The maximum allowable concentration of selenium compounds in air is 10 $\mu g/m^3$, which would yield an average daily intake level of 200 μg. Ziskind and Hausknecht (1976) note that no standards or regulations currently exist for inhalation exposure of the public to selenium, but that inhalation does not appear to contribute a significant quantity of selenium to the normal ingestion pathway. OSHA (1974) recommends 20 $\mu g/m^3$ for workers. This would yield a daily dose of 1200 μg, which is in excess of both typical dietary levels and Sakarai's recommended total daily intake level.

Figure 4.34 is a summary of much of the data given on exposure and health effects.

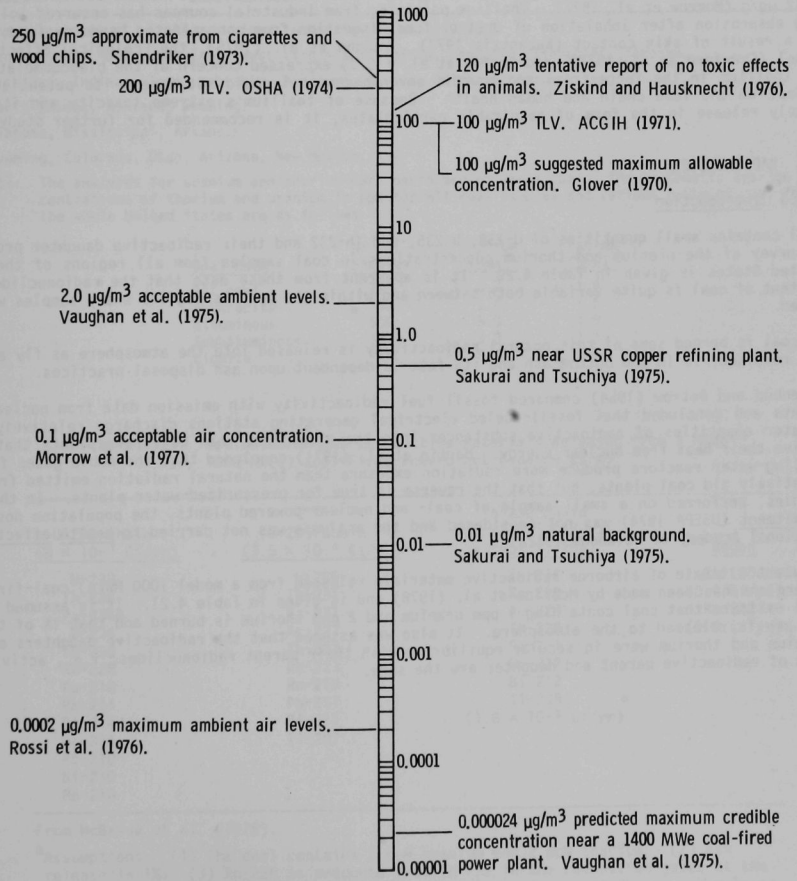


Fig. 4.34. Levels and Health Effects of Selenium (Se)

4.3.2.8 Thallium

Thallium (Tl) in coal is volatilized to the extent of 50% or more during combustion and released to the environment as particulates or gases (Heit 1977). It is more volatile in an oxide, sulfate, carbonate, silicate, or phosphate than in its elemental form and may be preferentially released to the atmosphere along with arsenic, cadmium, mercury and lead. Thallium oxide is believed to be the main species of thallium released by coal combustion (Beliles 1975). Natusch et al. (1974) also report that thallium, arsenic, antimony, cadmium, lead, and selenium are preferentially concentrated on particulates in the respirable range, a large part of which are not removed by control techniques.

Natusch et al. (1973) have measured thallium concentrations from 0.3 to 3 mg/kg in coal and from 50 to 90 mg/kg in airborne material collected within 100 m of coal combustion sources. Vaughn et al. (1975) predicted air concentrations of thallium of 3×10^{-6} $\mu g/m^3$ in the vicinity of a 1400-MWe coal-fired powerplant; Morrow et al. (1977) calculated an acceptable air concentration of 0.01 $\mu g/m^3$, comparing Vaughn's data to ambient air concentrations of 5×10^{-3} $\mu g/m^3$ (International Council on Radiological Protection 1975) and an occupational Theshold Limit Values (TLV) of 100 $\mu g/m^3$ (ACGIH 1971).

Thallium is cumulative in effect and quite toxic, with a lethal dose for humans estimated to be 8-12 $\mu g/g$ (Morrow et al. 1977). Thallium poisoning from industrial sources has occurred following absorption after inhalation of dust or fume, ingestion from contaminated hands or food, or as a result of skin contact (Kazantzis 1977). Vaughn et al. (1975), the Ecological Effects Panel (Crawford et al. 1977), and Morrow et al. (1977) expressed concern of the bioaccumulation of thallium in the terrestrial and aquatic environments but could not assess its potential effect on the food chain and human health. Because of thallium's extreme toxicity and its likely release in the form of respirable particulates, it is recommended for further study.

4.4 RADIONUCLIDES

4.4.1 Introduction

Coal contains small quantities of U-238, U-235, and Th-232 and their radioactive daughter products. A survey of the uranium and thorium concentrations in coal samples from all regions of the United States is given in Table 4.20. It is apparent from these data that the radionuclide content of coal is quite variable both between and within the various regions where samples were taken.

As coal is burned some of this natural radioactivity is released into the atmosphere as fly ash; the remainder is in the bottom ash and its fate is dependent upon ash disposal practices.

Eisenbud and Petrow (1964) compared fossil fuel radioactivity with emission data from nuclear plants and concluded that fossil-fueled electrical generating stations discharge relatively greater quantities of radioactive substances into the atmosphere than many powerplants that derive their heat from nuclear energy. Martin et al. (1971) concluded that the noble gases from boiling-water reactors produce more radiation exposure than the natural radiation emitted from relatively old coal plants, but that the reverse is true for pressurized-water plants. In these studies, performed on a small sample of coal- and nuclear-powered plants, the population dose commitment (USEPA 1974) was not considered and the analysis was not carried to health effects (National Academy of Sciences 1972a).

A recent estimate of airborne radioactive materials released from a model 1000 MW(e) coal-fired powerplant has been made by McBride et al. (1978) and is given in Table 4.21. It is assumed in this estimate that coal containing 1 ppm uranium and 2 ppm thorium is burned and that 1% of the fly ash is released to the atmosphere. It also was assumed that the radioactive daughters of uranium and thorium were in secular equilibrium with their parent radionuclides; i.e., activities of radioactive parent and daughter are the same.

Table 4.20. Range of Uranium and Thorium Concentrations and Geometric Means (expected values) for Coal Samples Taken from Various Regions of the United States

Region	Coal Rank	Number of Samples	Uranium Concentration (ppm) Range	Uranium Concentration (ppm) Geometric Mean	Thorium Concentration (ppm) Range	Thorium Concentration (ppm) Geometric Mean
Pennsylvania	Anthracite	53	0.3-25.2	1.2	2.8-14.4	4.7
Appalachia[a]	Bituminous	331	< 0.2-10.5	1.0	2.2-47.8	2.8
Interior[b]	Bituminous	143	0.2-43	1.4	< 3 -79	1.6
Northern Great Plains[c]	Subbituminous, lignite	93	< 0.2-2.9	0.7	< 2.0-8.0	2.4
Gulf[d]	Lignite	34	0.5-16.7	2.4	< 3.0-28.4	3.0
Rocky Mountain[e]	Bituminous, subbituminous	134	< 0.2-23.8	0.8	< 3.0-34.8	2.0
Alaska	Subbituminous	18	0.4-5.2	1.0	< 3.0-18	3.1

From Swanson et al. (1976) as cited in McBride et al. (1978).

[a]Pennsylvania, Ohio, Maryland, West Virginia, Virginia, Kentucky, Tennessee, Alabama.

[b]Michigan, Indiana, Iowa, Nebraska, Missouri, Kansas, Oklahoma, Arkansas.

[c]North Dakota, Montana, Wyoming.

[d]Alabama, Mississippi, Arkansas.

[e]Wyoming, Colorado, Utah, Arizona, New Mexico.

Note: The analyses for uranium and thorium were performed on whole coal. The arithmetic average concentrations of thorium and uranium in ppm for all coal samples and various ranks of coal for the whole United States are as follows:

Coal Rank	No. of Samples	Thorium (ppm)	Uranium (ppm)
All coal	799	4.7	1.8
Anthracite	53	5.4	1.5
Bituminous	509	5.0	1.9
Subbituminous	183	3.3	1.3
Lignite	54	6.3	2.5

Table 4.21. Estimated Annual Radioactive Materials Released from a Model 1000-MW(e) Coal-Fired Powerplant (source term)[a]

U-238 chain (8×10^{-3} Ci/yr)	U-235 chain (3.5×10^{-4} Ci/yr)	Th-232 chain (5×10^{-3} Ci/yr) except Tl-208)	Radon
U-238	U-235	Th-232	Rn-220
Th-234	Th-231	Ra-228	(0.4 Ci/yr)
Pa-234m	Pa-231	Ac-228	Rn-222
U-234	Ac-227	Th-228	(0.8 Ci/yr)
Th-230	Th-227	Ra-224	
Ra-226	Ra-223	Pb-212	
Po-218	Rn-219	Bi-212	
Pb-214	Pb-211	Tl-208	
Bi-214	Bi-211	(1.8×10^{-3} Ci/yr)	
Po-214	Tl-207		
Pb-210			
Bi-210			
Po-210			

From McBride et al. (1978).

[a]Assumptions: (1) The coal contains 1 ppm uranium and 2 ppm thorium. (2) Ash release is 1%. (3) Rn-220 is produced from Th-232 in the combustion gases at the rate of 1.38×10^{-9} curies per second per gram of thorium. (4) The annual release of natural uranium is 2.32×10^{4} g and of Th-232 is 4.64×10^{4} g. (5) 15 sec are required for the gases to travel from the combustion chamber to the top of the stack.

4.4.2 Summary of Health Effects

Exposure to radioactivity in fly ash is mainly by inhalation of small particles, ingestion of contaminated terrestrial foods, and direct radiation from deposition on ground surfaces. The ingestion pathway represents the largest potential exposure mode, but this pathway is highly dependent upon the quantity of agricultural crops growing in the environs of a coal-fired plant. Radiation doses and exposure pathways from the study by McBride et al. (1978) are given in Table 4.22. The ingestion doses estimated in this study are based on an assumption that all food is grown and consumed in the study area (area within 80 km of the plant). Therefore, coal-fired plants in urban areas with little agriculture could be expected to have less exposure via ingestion of contaminated food. The dose due to inhalation would be the major pathway of exposure. However, more people may live in the urban setting so that the population dose (man-rem) could be as high as when ingestion pathways are significant. In any case, population doses are very similar to those for persons exposed to radiation from routine releases from nuclear powerplants. Moreover, the combustion of coal containing <1 ppm uranium and 2 ppm thorium or fly ash treatment systems that release more than 1% of the ash could be expected to lead to higher radiation doses than those cited here. Additional exposure would be expected from the bottom ash, depending upon the fate of this material, which may be treated as waste or used for construction.

Maximum individual dose commitments from the airborne releases of model 1000-MWe powerplants are compared with Code of Federal Regulations (CFR) guides in Table 4.23. The 50-year dose commitment from the annual airborne releases from both the model coal-fired and nuclear plants should be viewed in the perspective of the annual dose resulting from natural background radioactivity (130 mrem/yr; external plus internal radiation, in the U.S.).

McBride et al. (1978) consider the public health significance of the estimated dose commitments for the model plants as relatively minor. Using an estimate of 100-200 health effects (i.e., cancer mortality and genetic defects in the first two generations following exposure) per 10^6 whole-body man-rems (National Academy of Sciences 1972a), a rate of 0.001-0.003 health effect per year of operation of the model nuclear plant is estimated, compared with 0.002-0.005 health effect per year of operation for the model coal-fired plant.

Teknekron (1977) also performed a health effects assessment for radionuclides emitted to the atmosphere from burning Appalachian coal. Their results are summarized in Table 4.24.

Using the value of ~0.1 excess cancers per year per typical 1000-MWe coal-fired generating station as a reasonably valid health effect estimate on a nationwide basis, Teknekron derived a health effect impact estimate for current utility coal-burning operations. The 1974 national coal-generated electricity output of 0.830 trillion kWh is roughly equivalent to the output of 120 of the model 1000-MWe generating plants. The estimated national health impact resulting from radionuclide emissions by current (1974) electric generation from coal is, then, 12 excess cancers per year. Recognizing the uncertainties imparted by the assumptions that are necessarily included in this type of estimate, Teknekron concluded that the health impact resulting from radionuclide emissions by coal-fired generating stations are insignificant as compared to known and suspected health effects resulting from other coal-derived pollutants.

Table 4.22. Radiation Doses and Pathways of Exposure from a Model 1000-MWe Coal-Fired Powerplant

Organ	Individual[a] (mrem/yr)	Population[b] (man-rem)	Percent Contribution to Dose[c]		
			Ingestion	Inhalation	Surface
Whole body	1.9	21	93.6	5.5	0.9
Bone	18.2	225	82.9	17.0	0.1
Lungs	1.9	29	62.4	37.2	0.4
Kidneys	3.4	50	85.4	14.4	0.2
Liver	2.4	29	93.1	6.5	0.4

From McBride et al. (1978).

[a]Maximum dose to individual at 0.5 km distance.

[b]Dose to 3.5 million persons living within 80 km of the release point (100-m stack).

[c]Dose to population.

Table 4.23. Maximum Individual Dose Commitments from the Airborne Releases of Model 1000 MWe Powerplants[a] Compared with Code of Federal Regulations (CFR) Guides (10)

Organ	Maximum Individual Dose Commitment (mrem/year)			
	Coal-Fired Plant[b]	BWR[c]	PWR[c]	CFR Guide[d]
Whole body	1.9	4.6	1.8	5
Bone	18.2	5.9	2.7	15
Lungs	1.9	4.0	1.2	15
Thyroid	1.9	36.9[e]	3.8	15
Kidneys	3.4	3.4	1.3	15
Liver	2.4	3.7	1.3	15
Spleen	2.7	3.7	1.1	15

[a]The maximum individual dose commitments are for a midwestern site and are estimated at the plant boundary at 500 m from the release points. Dose commitments are less at greater distances. The ingestion component of the dose commitment is based on the assumption that all food is grown and consumed at the reference location.

[b]The dose commitments listed are essentially the same for all stack heights 50 to 300 m, including the plume rises resulting from buoyancy of hot stack emissions, A 1% ash release was assumed. The coal was assumed to contain 1 ppm U and 2 ppm Th.

[c]Source terms for the nuclear plants are from U.S. Nuclear Regulatory Commission (1976). The release height was assumed to be 20 m with no plume rise.

[d]Except for whole body, values given are design guides for doses from iodine and particulates.

[e]Assumes dairy cow on pasture at site boundary for entire year. The thyroid dose estimated (20, p. IV C-115) for the same source term was 11.7 mrem/year. The lower number results from the assumption that the dairy cow is on pasture only a fraction of a year.

Table 4.24. Results of Health Effects Assessment for Radionuclides Emitted to the Atmosphere from Burning Appalachian Coal[a]

Radionuclide	Assuming Soluble Particles		Assuming Insoluble Particles	
	Bone Dose (man-rem)	Health Effects (excess cancers)	Lung Dose (man-rem)	Health Effects (excess cancers)
Uranium chain				
Ra-226	1,240		320	
Th-230	550		320	
Subtotal	1,790	~0.02	640	~0.03
Thorium chain				
Ra-228	8,580		1,330	
Th-228	250		370	
Th-232	1,250		210	
Subtotal	10,100	~0.12	1,910	~0.08
Total	11,900	~0.14	2,550	~0.11

From Teknekron (1977).

[a]1000-MWe generating plant operating at 80% load factor in area with 160 people per square mile.

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